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XLIX.

# VITAMINS IN HUMAN AND ANIMAL NUTRITION.*

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One of the first researches, which led, eventually, to the vitamin hypothesis, was that of Lunin, who published a paper in 1881 stating that he had fed purified rations (to experimental animals) which were incapable of producing growth. The addition of a small amount of milk, however, caused the animals to resume their normal rate of growth. This paper was published in a journal of limited circulation, with the result that Lunin's observation was not brought to the attention of scientists for a number of years.

Probably the first piece of work to really stimulate interest in this field was that of Eijkmann (1897), a Dutch physician, who produced experimental Avian beri-beri by feeding polished rice to poultry. Eijkmann was able to show that the

*Presented before the Fifty-first Annual Congress of the American Laryngological Association, Atlantic City, May, 1929, as part of a symposium on "Nutritional Disturbances in Relation to the Nose."

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polishings or branny portion possessed curative properties and, later, he succeeded in curing and preventing this deficiency disease by feeding or injecting an extract of rice polishings. He concluded that white rice contained a toxin or poison that was the prime etiologic factor in beri-beri, and he postulated that the curative properties of the polishings were due to the presence of a substance which acted as an antidote. In spite of this incorrect conclusion, his work attracted considerable attention and stimulated further work by other investigators.

Another important series of observations that paved the way for vitamin research were those of Hopkins of England. Dr. Hopkins showed, as early as 1906, that as little as 2 cc. of cow's milk caused rats to grow on purified diets. He pointed out that this stimulation could not be due to fats, proteins, carbohydrates or mineral salts. He postulated that milk must contain some hitherto unknown "accessory food factor." He is given the credit, therefore, for suggesting the existence of these food factors that we now call vitamins.

Between the years 1910 and 1912 Dr. Casimir Funk repeated and substantiated the work of Eijkmann. Being a chemist, however, he pursued the problem further, with the result that he was able to isolate from rice polishings a crystalline chemical substance which contained carbon, hydrogen, oxygen and nitrogen. This substance was curative in avian beri-beri and possessed basic properties. As a result, Funk suggested the name "vitamine" for this food factor. Some years later the "e" was dropped from the word "vitamine" in order that this class of substances should not be confused with the plant alkaloids such as strychnin, morphin, etc.

The first systematic work in vitamin research in this country developed in the laboratories of Osborne and Mendel at Yale and in the laboratories of Babcock, Hart and McCollum at Wisconsin. It was not long before these laboratories obtained proof for the existence of a fat-soluble food factor in cod liver oil, butter fat and egg yolk. The absence of this factor from the diet caused loss in weight, accompanied by the development of xerophthalmia. At about the same time McCollum and Kennedy showed that the antineuritic factor of Funk possessed growth promoting properties when fed to

rats. McCollum and his co-workers were unable to find that the fat-soluble factor contained nitrogen and for that reason refused to adopt the term "vitamine." They suggested, therefore, that the fat-soluble factor be known as "Fat-soluble A" and that the water-soluble factor in rice polishings and in yeast be called "Water-soluble B."

Lack of time prevents further discussion of the discovery of the various vitamins. I wish to say, however, that the term vitamin appealed to popular and scientific fancy, with the result that it has become official and universal in usage.

Then came the discovery of vitamin C, the antiscorbutic factor, which was found in citrus fruits, sprouting grains, green leafy plants and in many vegetables and fruits.

For a time the work of Mellanby and others seemed to indicate that vitamin A also possessed antiratchitic properties. Mellanby also noted that dogs developed rickets in confinement much more rapidly than when they were allowed to run out of doors. At that time he was inclined to believe that exercise was curative or at least preventive in rickets. Later work on heliotherapy, however, has provided an explanation for his results.

McCollum and co-workers were able to destroy vitamin A in cod liver oil without injuring the antiratchitic potency of the oil appreciably. This work led to the introduction of the term "vitamin D" to designate the antirachitic factor.

A few years later Dr. Herbert Evans of California introduced proof for the existence of an antisterility factor which he first called "vitamin X," but later the term "Vitamin E" was adopted. This factor, according to Evans, brings about fertility when rats have been made temporarily sterile on certain types of rations. He finds this factor quite universally distributed, but finds it in high concentration in cereal oils, particularly wheat germ oil. This factor is capable of withstanding saponification and distillation.

Recently the California workers have suggested a new factor called "vitamin F," but since some investigators are questioning its existence I shall not take time to discuss it.

Within the past few years evidence has accumulated to prove that the old vitamin B is really a "complex," consist-

ing of at least two, and possibly more factors. As a result vitamin workers have given scientific recognition to two fractions. The first one, which cures beri-beri and which is heat labile is now called "vitamin B" by American workers and "vitamin $B_1$" by British workers. The second fraction of the old vitamin B complex, which promotes appetite, cures skin lesions (pellagra) and which is thermo-stable, is now called "vitamin G" by American workers and "vitamin $B_2$" by British workers.

Vitamin A is found in the unsaponifiable portion of those fats which contain it. It is fairly stable to heat in the absence of oxygen, but in the presence of oxygen it is destroyed. Even in the absence of heat it is susceptible to slow oxidation. In a general way the vitamin A content of foods is related to pigmentation—i. e., those foods which are rich in the carotinoid pigment are usually rich in vitamin A. There are exceptions to this rule, however.

Vitamin B, the antineuritic factor, is soluble in water and water-alcohol solutions. It can be precipitated, dialyzed, and adsorbed on the surface of colloids. It is quite easily destroyed by heat as compared with vitamin G.

Vitamin C, the antiscorbutic factor, is also water-soluble, dialyzable and is stable in acid solutions but destroyed by alkalies. It is also very susceptible to oxidation, but may be considered fairly stable to heat if oxygen is absent.

Vitamin D, the antirachitic factor, is quite stable to heat, is soluble in the fat solvents, is stable to saponification reactions and is associated with the sterol fraction of those fats containing it. Recent work indicates that the alcohol "ergosterol" is identical with or is the parent substance of vitamin D.

Vitamin E, the antisterility factor, as I have already indicated, is fat-soluble, heat stable and is found in the unsaponifiable fraction of wheat germ oil.

Vitamin G, the appetite promoting, pellagra preventive factor, is soluble in water and water-alcohol solutions. It is not absorbed at the same hydrogen-ion concentration as the antineuritic factor, and from its deportment it is thought that it is a nitrogenous compound also. Much of the old work on vitamin B must now be repeated, before we can interpret all data correctly.

MEDICAL AND NUTRITIONAL ASPECTS.

May I explain that the literature is full of ideas and opinions, many of which are unsupported by experimental evidence, relative to the physiologic function of vitamins? I shall attempt to mention but a few.

Functions of Vitamin A.—Some writers believe that vitamin A promotes the function of tissues and that blood platelets fluctuate with the supply of this food factor. It is certain that appetite, growth and well-being are dependent on this vitamin. A deficiency of vitamin A causes loss of appetite, loss in body weight and susceptibility to infection, particularly of the eyes, ears, sinuses, air passages and lungs. In the advanced stages of vitamin A deficiency we find xerosis, corneal ulcers, colds, influenza, pneumonia, otitis media and, occasionally, infections at the base of the tongue. Renal calculi have also been noted in many cases. Diets containing adequate amounts of cod liver oil, butter fat, cream, carrots, liver, etc., prevent the pathologic conditions just described.

Functions of the Vitamin B Complex.—Owing to our lack of knowledge regarding the new factors B (or $B_1$) and G (or $B_2$), I shall discuss them together. When the complex is omitted from the diet appetite fails and losses in body weight and emaciation ensue. Death may occur with or without the development of polyneuritic symptoms. Usually, the animals are constipated and are characterized by marked loss in vigor.

In all stages of vitamin deficiency there is a characteristic loss of sexual interest and, consequently, failure to breed. My own work and that of other investigators shows that there is a tendency for all vital organs of the body to atrophy in vitamin B (complex) deficiency with the exception of the suprarenal glands, which show a distinct tendency to hypertrophy.

There seems to be some relationship between nuclear nutrition and vitamin B (complex) supply. McCarrison has been so positive of this that he has suggested that we adopt the term "nucleopast" to emphasize that these factors stimulate nuclear metabolism. I have already noted that the absence of vitamin B ($B_1$) induces polyneuritis and that vitamin G ($B_2$) has to do with appetite stimulation and the prevention of

pellagra. Our own researches have indicated that this complex may function, directly or indirectly, as a stimulator of the oxidative processes, but this theory has been questioned by some investigators.

The vitamin B complex may be obtained from yeast, whole grains, vegetables, egg yolk, fruits, leafy vegetables and milk.

Functions of Vitamin C.—This is the antiscorbutic factor and, while necessary for many animals, does not seem to be necessary for rats or poultry. Guinea pigs are very susceptible to diets deficient in this factor. In the guinea pig we find that the functions of vitamin C and the pathology of scurvy are similar to if not identical with those observed in human scurvy. When the diet is deficient in this factor we note first loss in appetite and body weight, followed by weakness, rapid respiration, abnormal posture, swollen and tender joints, hemorrhagic gums, loss of teeth and paralysis. Postmortem examination reveals muscular hemorrhages, hypertrophy of adrenals and friable bones.

Orange juice, lemon juice, tomato juice, sprouted grains, leafy vegetables and fruits are good sources of vitamin C.

Functions of Vitamin D.—This is the antirachitic factor which plays a part in controlling calcium-phosphorus equilibrium and deposition. The outstanding symptom of vitamin D deficiency is the softness of bones and teeth. The ratio of calcium to phosphorus in the diet is a most important factor. Calcification is enhanced under all conditions, however, by vitamin D.

Recent work by Windaus, Rosenheim and Webster, Hess, Steenbock and others has led to the conviction that the alcohol ergosterol is the parent substance or precursor of vitamin D. The treatment of inactive ergosterol with ultraviolet light produces an antirachitic compound which is at least 100,000 times more potent than cod liver oil. The potency of antirachitic agents may be studied by four methods—i. e., (1) X-ray, (2) line test of bones, (3) blood phosphorus, or (4) mineral deposition, as measured by chemical analysis of the bone.

I shall not discuss the pathology of nutritional sterility and vitamin E function, owing to the lack of time, except to say that Evans and his co-workers describe several causes of

sterility, such as (1) failure of fertilization, (2) fertilization takes place but implantation does not, (3) fertilization and implantation occur but the fetus dies and is resorbed.

At this point Professor Dutcher showed about 30 lantern slides descriptive of the various types of vitamin research in his own and in other laboratories. He described his own work showing how it was discovered that the vitamin content of milk is dependent on the vitamin content of the diet. He described recent experiments in which he has found that rats cannot utilize vitamin A when butter fat is fed in juxtaposition to mineral oil or liquid petrolatum. He explained that this was not due to increased intestinal motility accompanied by rapid bowel evacuation. Neither did it appear to be a matter of intestinal permeability. He expressed the belief that it is most likely due to solubility of Vitamin A in the mineral oil, which is not assimilated.

Professor Dutcher also described the cooperative research of Bechdel, Dutcher and Knutsen in which they proved that the dairy cow could live, grow and reproduce on diets deficient in the Vitamin B complex. Subsequent research by means of a gastric (rumen) fistula proved that the cow is capable of developing bacteria in the rumen which synthesize sufficient vitamin B complex for her needs. An organism was isolated which caused rats to grow on diets deficient in the B complex. This organism, which had not been described, has been named "Bacterium Vitarumen".

In closing, he discussed ultra violet light and food irradiation and how we should make practical applications of our knowledge. He summarized his discussion by stating that while deficiency diseases are existent and important, that probably our main problem was not to worry about the development of deficiency diseases but rather to try to feed sufficiently varied diets to prevent lowered resistance and susceptibility to other diseases. He stated that, in the great majority of cases, it was possible and preferable to obtain our vitamin supply from the dairy, the garden, the orchard and the poultry yard.

L.

# DIET IN CONNECTION WITH DISEASES OF THE LYMPHATIC SYSTEM, ESPECIALLY OF THE UPPER AIR TRACT.*

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The lymphatic system is made up of capillaries and vessels connecting a complicated network of nodules or glands. Much has been written on the changes of the lymphatics due to infection, but there is a great paucity of literature dealing with the effect of diet upon their behavior. In this day and age, where so much work has been done relative to dietary effects on diseased and normal tissue, it seems timely to consider whether or not infection, especially of the respiratory tract, may not be encouraged if not actually invited by changes brought about by an improper balance in the diet of the organism.

We know that the lymphatic glands are actively concerned with the arresting and walling off of infection, and if an otherwise healthy gland is so altered by dietary disturbances, knowledge of this fact will enable us to aid nature in combating the attack of what may prove to be an unfavorable termination of one of the many manifestations of respiratory infection.

Blumgart reports autopsies on three cases of malabsorption of fats in which he noted some lymphatic disturbances. These were patients brought to the hospital complaining of progressive emaciation in spite of all efforts to arrest the same. The emaciation or wasting was accompanied by absorption of subcutaneous and tissue fat. The feeding of fat in the diet was without effect, and laboratory tests showed large quantities of fat in the stool. The cases were all fatal, and autopsies

*Presented before the Fifty-first Annual Congress of the American Laryngological Association, Atlantic City, May, 1929, as part of a symposium on "Nutritional Disturbances in Relation to the Nose."

showed thinning of all tissues together with degeneration. The lymphatics—especially abdominal lymph glands—were slightly enlarged and one section showed hyperplasia and edema and occasional cloudy swelling. No particular mention was made of the lymphatics of the upper air tract, but we might be justified in assuming that the same changes exist there.

Jackson reports that, "Although the characteristic involution of the lymphoid tissue during malnutrition usually results in atrophy of the lymph nodes, they often appear swollen (especially in rickets, beriberi and scurvy), perhaps chiefly through secondary infections in conditions of lowered resistance.

During total inanition the quantity of lymph apparently increases during the first third of the fasting period, but gradually decreases later, with changes in composition.

The lymphatic glands during inanition appear variable in size. In emaciated human adults they are in many cases extremely atrophic, but in others they appear normal in size or even enlarged (probably from secondary infections). Enlargement appears more frequent in atrophic infants. In fasting animals, the results are also variable, although marked atrophy of the lymph glands appears characteristic. During partial inanition, changes in the size of the lymph glands likewise appear somewhat constant, but enlargement appears characteristic in rickets, beriberi and scurvy, especially in the mesenteric nodes, often probably due to secondary infection.

Microscopically, the lymphatic glands during inanition usually show a very characteristic atrophy of the lymphoid tissue, even in cases where a decrease in the size of the gland as a whole may be offset by a distension of the blood vessels and lymph sinuses. In general, there is a marked diminution in the number of lymphocytes (by emigration), which renders the less affected stroma (reticulum) and trabeculæ very prominent. The lymphoid nodules and cords are reduced in size, and mitoses are decreased in number or absent. Numerous phagocytic cells are found, often containing pigment derived from excessive destruction of erythrocytes (especially in regions of hemorrhage in scurvy). An increased number of phagocytes and plasma cells has been noted during hibernation. Reterer's claim that lymphatic glands may be transformed into hemolymph glands by inanition lacks confirma-

tion. Secondary infections may occasion inflammation, however, and occasionally even suppuration of the lymphatic glands, especially in scurvy.

Cirrhosis of the lymph glands has been noted in pellagra. The lymphoid tissue appears especially sensitive to a dietary deficiency of fat, while in rickets a general lymphoid hyperplasia appears characteristic. A deficiency in vitamins (especially of vitamin B) tends to cause a general atrophy of the lymphoid tissue, associated with lymphopenia in the circulating blood. During chronic thirst, the changes in the lymph nodes resemble those typical for inanition in general, with hyperemia and lymphoid strophy.

Although there are numerous variations, the changes in the structure of the lymphatic glands during inanition in general resemble those found in the other lymphoid organs, including the bursa of Fabricius ("cloacal thymus" in birds), bone marrow (considered with the skeleton) and the thymus spleen and intestinal lymphoid structures.

Kohman found congestion of the lymph glands in rats with edema produced by diets deficient in protein and fats. Settle noted hypertrophy of lymphoid organs of kittens on a diet rich in fat and high in calories.

With this amount of information at hand, I examined albino rats which had been fed upon a normal diet. Autopsies on the normal rats showed nothing microscopically, as the lymphatics are tiny. Therefore, masses of tissue in the bronchial lymph node region were embedded in paraffin and sectioned. This gave us an idea of the natural structure and appearance of the lymphatic glands, for embedded in the loose areolar tissue could be seen here and there lymphatic tissue.

A batch of young rats were then fed upon a diet free from vitamin A or fat soluble A. These animals soon developed the characteristic changes as I have reported in previous articles. There appeared a marked conjunctivitis—watery secretion from the nose and in some cases corneal ulcers. Autopsies on these animals revealed very little demonstrable change in the bronchial lymphatics. I thought in some instances there was the suggestion of cloudiness supporting the theory of

edema or hyperplasia, but this was so slight and so inconstant that I did not feel justified in accepting it as an unchallenged fact.

It is well known and generally accepted that diet free of vitamin A predisposes to sinusitis, bronchitis and pneumonia. The mucosa of the respiratory tissue is so altered that secondary infection is bound to set in. If, however, in animals dying during this type of experimentation, we find lymphatic enlargement, we are not justified in attributing the lymphatic involvement to the diet—we must assume that the swelling is due to the toxins and reaction of infection.

Animals fed upon diets deficient in vitamins B and C showed no lymphatic changes. A vitamin B deficient diet is one producing changes similar to the appearance of starvation. McGarrison reports extensive changes in all tissues of patients dying of beriberi in India, but has noted only slight enlargement of the abdominal lymphatics, and this was probably relative rather than actual. But here again, as is also true in pellagra, it is a question of secondary infection, and it is almost impossible to say just where the influence of nutritional disturbances ceases and the action of toxins from the infection begins. I then examined rats which had been fed on a rachitic diet—when vitamin D was eliminated from the diet. These animals were in advanced stages, waddling gait, roughened coat, stupid and all of the characteristics of the disease. Examination showed no change or enlargement of the lymphatics.

At this point, therefore, we might say that in the light of the experiment thus carried out a diet defective in vitamin has little or no effect in itself upon the condition of the lymphatic system. The questionable changes in cases deficient in vitamin A are not conclusive enough to go unchallenged. There can be no doubt that vitamin A deficiency probably renders the lymph glands susceptible to infection just as it causes edema and lowered surface tension in the respiratory mucosa, but we are unable to identify this in the rats. This, of course, renders all that area prone to bacterial invasion. The infection takes place so early that we are forced to conclude that enlargement, if any, is due to bacterial invasion

rather than diet. It will be of interest to carry on this work using larger animals, such as rabbits or a dog, but in the time allotted for this work this was impossible at this time. Larger animals develop tissue changes slowly, and consequently changes noted must necessarily be of slight degree.

We then felt that possibly some light might be thrown upon the subject by studying fat metabolism. A large dog was starved for several days. Twenty-four hours before we were to examine him we fed him large quantities of fat which had been stained with Sudan III dye. The object was to render the lymphatic system impoverished of fats and then by rapid ingestion of the stained fat to load the tissues. It was thought possible to dissect and trace the lymphatics of the upper air tract in that way. This was not satisfactory.

In the course of some of the experimental work which I continued one dog failed to have supplied to him the normal amount of drinking water while he was on a fat free diet. This animal showed definite changes of all lymphatic tissue in the nature of softening and hyperemia or congestion.

It would appear then that if any one element in diet has a harmful reaction in the lymphatic system it is the fats, and this may be made even more so by the deprivation of water. This relationship of food and water has been demonstrated by Newberg, who found that rats and dogs fed on a diet high in protein and free from fat—such as dried liver—developed kidney changes more rapidly and extensively when water was restricted than otherwise.

In considering the diets of various tribes of people living in isolated lands we are struck by the fact that the Eskimo lives on a diet high in fat content, and the native of India, quite the opposite, lives on a low fat diet. These comparisons do not check exactly with laboratory animal experimentation, but we can at least claim that laboratory work points out the trend or tendency.

Animals fed upon a diet rich in sugars demonstrated no change in the respiratory lymphatics. In the main the sweets seemed to inhibit the appetite and they refused to eat the ordinary fare.

The analysis of our diets during the world war while we were restricted in the use of sugars is interesting in that respiratory diseases were very prevalent. However, it would be impossible to draw definite conclusions, as the respiratory infection itself would cause enlargement of the respiratory lymph nodes.

The practical application of this work then would point to the administration of fats, in any form, and plenty of water or fluids for a patient suffering from a respiratory infection or for a well balanced prophylactic diet. This, after all, only substantiates a time honored form of therapy in the face of such findings.

In conclusion, I would say that so far nothing definite can be claimed as to the effect of diet upon the respiratory lymphatics. The weight of evidence rather points to the probability of fat being of clinical value—that is, fat per se is essential in the diet. Vitamins themselves probably are not responsible for lymphatic changes, except vitamin A, and, as said before, it is difficult to say how much of the lymphatic enlargement is due to vitamin A deficiency and how much to secondary infection. It is hoped that further experimentation will continue and more data added to our present knowledge.

Sugars did not seem to alter the lymphatics. This in a way is a preliminary report. There is a great deal of material collected under the classification of diets, and one would gather the impression that the laboratory findings had solved the problem of respiratory infection. On the contrary, far from it. It must be remembered that man is an organism made up of simple cells which are prone to invasion by outside influences, bacterial, chemical, thermal, etc., and that all experimental work on diets is an effort to render the single cell an unfavorable growing media for bacteria.

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LI.

# THE RELATION OF DEFICIENCY DIET TO DISEASES OF THE SINUSES.*

By Lee Wallace Dean, M. D.,†

St. Louis.

About 1918 Dr. Amy Daniels demonstrated the relationship between sinus infection and diets deficient in the fat soluble vitamin A. At that time there was no question as to the influence of diet upon sinus disease. There was some question as to whether dietary treatment should supersede surgical or other treatment of chronic sinus infection.

To help clarify the situation, Dr. Byfield and Dr. Daniels selected a child, seven years of age, suffering from chronic nasal sinus disease, and by hospitalization with dietary and other forms of general treatment attempted to combat the infection. After six weeks of effort there was no change in the general condition of the child. It remained undernourished, underweight, and its appearance was very poor.

At the end of this time the child was transferred to the laryngological service to see what could be gained by treatment of the sinuses. With drainage and other laryngologic treatment of the nasal condition the child immediately improved. Then Dr. Daniels added dietary treatment to the laryngologic and the child improved very much more rapidly.

It is to be noted in this connection that the use of vitamins did not improve the child: a much more beneficial result was secured by drainage and treatment with an ordinary diet than by diet and general treatment.

The first thing to be kept in mind in connection with the treatment of chronic nasal sinus disease in children is that if the best result is to be secured the proper laryngologic

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treatment must accompany the dietary. In this matter Dr. Daniels is in complete agreement and for years, by working together, we demonstrated to our satisfaction the beneficial results of teamwork in this class of cases.

Only in incipient nasal infections the desired result may be secured by dietetic and hygienic treatment alone.

Baby Sammy, seven months old, in Dr. Daniels' service, developed a nasal infection with fever and loss of weight. With a diet rich in vitamin A he did not make satisfactory progress, and Dr. Daniels was about to telephone me when she decided to add orange juice and large amounts of vitamin B, secured from wheat kernels, to the diet. This brought about the desired effect. Dr. Daniels thinks that perhaps the administration of vitamin B may increase the absorption of vitamin A from the intestinal tract. This and many other cases have demonstrated the fact that in acute nasal infections in children diet with proper hygienic and systemic measures may bring about a satisfactory result even when acute sinus trouble is present. At any rate, in sinus infections the proper administration of vitamins is a most important part of the treatment. Of equal importance is the proper hygienic conditions.

Whenever there is interference with the drainage of the sinuses it is questionable as to the amount of good that will be secured by the administration of vitamins. Deficient drainage should always be corrected.

In adults as well as children a diet rich in vitamin A is indicated. The influence of deficient diets is not so marked in adults as in children. Marriott[1] suggests that this is due to a fair amount of vitamin A being stored in the body in glandular fats. He remarks, however, that ultimately, whether young or old, individuals deprived of vitamin A develop loss of strength and resistance to infection, especially tuberculosis and sinus infections.

About 1918, Dr. Daniels asked Dr. Armstrong to autopsy a white rat in which an ophthalmia had been produced by the feeding of a diet deficient in vitamin A. Dr. Armstrong investigated the nasal sinuses and found a suppurative sinusitis which was not present in the control rat. Drs. Daniels and Armstrong carried on a series of observations. They showed that the microscopic changes in the infected rat were similar

to the human. Dr. Daniels showed that the sinus infection could always be produced by a diet deficient in vitamin A.

At a meeting of the middle section of the Triological she brought before our group rats fed on a deficient diet and control rats. They were autopsied by visiting laryngologists with findings exactly as described.

Dr. Daniels later demonstrated that in the early stages of sinus infection in the rat, produced by deficient diet, the sinusitis could be corrected by suitable diet: that when once established, diet would not bring about a cure. This confirmed the observations that we had made by study of our patients. Following this observation we established a normal infant ward to study the growing infant.

Dr. Daniels was placed in charge. Newborn infants were placed in the ward and remained there until they were two years old. We must remember that not only did these infants receive a proper diet, so far as vitamins were concerned, but the inorganic substances were properly balanced. There were ideal hygienic surroundings; there was a minimum opportunity for infection. During the two years neither a laryngologist nor an otologist was summoned to the ward. Incipient nasal infections were controlled. When these children left, at the age of two years, they were kept under supervision in their adopted homes. One only developed a mastoid. In this case it was found that the mother desired to do things just right, so she mixed the baby's milk with cod liver oil, allowed it to stand and then skimmed off the surface content. Removing the fat resulted in a mastoid. At the age of seven none of these children have a caries tooth.

Diets deficient in vitamin A predispose infections. These infections are not limited to the nasal sinuses. The result is a nutritive disaster.[2]

Lambert and Judkin[3] studied infections in other parts of the body.

Emmett[4] found that in rats fed on diets deficient in vitamin A, with the exception of specific pathologic changes in eye tissue, there were only a few pathologic findings in other tissues. Haladay[5] observed that the nasal passages became involved, and a characteristic respiratory infection usually appeared in the advanced stages of the eye infection. Rats on

low fat soluble A diets developed snuffles, and with change of diet the snuffles disappeared.

In dissecting skulls of animals with eye infections produced by diets deficient in the fat soluble vitamin it was observed that in all cases the paranasal sinuses and mastoid cells contained purulent material. These findings led to suspect that in those animals receiving the low fat soluble A diet which had died from no perceptible cause, the infection was located in the upper respiratory passages.

Sixteen rats fed on low fat soluble vitamin diet for six to eight weeks developed snuffles and were rapidly losing weight; seven had lesions involving one or both eyes and several others had pigmented skin lesions on the eyelids and feet. These rats were killed. Microscopic examination of longitudinal section was made through the head, exposing the entire nasal mucosa; without a single exception the mucosa was found to be covered with a thick creamy exudate which filled the meatus.

Rats fed on low fat soluble vitamin developed snuffles; add cod liver oil and they responded immediately. Microscopic examination of the nasal mucosa of the animals confirmed the gross findings: the nasal sinuses filled with pus.

Bloch[6] has expressed the opinion that deficiency in vitamin A is clinically manifested not only by arrest of growth but by a decline in resistance to infection.

Sherman and Burtis[7] report that the level of intake of vitamin A during early life may markedly cause subsequent susceptibility to infection.

The absence of vitamin in the diet does not cause infection. From the time we are born until we die we combat the streptococcus. Early in life he is in the lymphoid tissue of the throat, then later in the sinuses, then in the gall bladder, the appendix, the teeth, then he lives in the prostate, the cervix of the uterus, in the colon. It is impossible to cut out all the places in which he lives. By keeping up our immunity, our resistance, or what not, we fight our battle best. Vitamins and proper hygienic measures are important things in this battle.

Perhaps in our climate they play a more important rôle than in the tropics. Dr. Lambert in a personal communication told

me that in Porto Rico a rickets commission found almost 100 per cent malnutrition in the children but only two cases of rickets. One of these was from New York, the other has always lived in a basement. It is the deficiency of vitamins plus something else that causes the sinus infection.

In the treatment of sinus infection a diet rich in vitamin A together with an increase in vitamin B is very essential. It, however, does not take the place of either the proper laryngologic procedures or the observance of the usual hygienic regulations. The latter two things are equally important.

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LII.

# DEFICIENCY DIET IN RELATION TO THE SKELETON, ESPECIALLY IN CONNECTION WITH THE BONE AFFECTIONS OF THE HEAD.*

By B. R. Shurly, M. D.,

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The skeletal structure in general, and the bones of the head and face in particular, have been of special interest to the otolaryngologist. In endeavoring to relieve the various pathologic findings and abnormalities we are struck by the fact that man is becoming with great rapidity a more pronounced artificial animal. Many of our surgical problems are developed as a result of lymphoid or bony overgrowth and resulting obstruction.

Why are the nasal passages, with their importance in the preservation of health, so frequently deformed and hypertrophied? If we may enter the field of preventive otolaryngology with the same scientific zeal attained in preventive medicine in general, we are aware that the future realm of scientific value to humanity is not in surgery, medicine or otolaryngology but in research study directed along the lines of biochemistry that will solve the problems of infection, immunization, the rôle of the endocrines, and the chemistry of foods. The untold prophylactic value of scientific feeding, the maintenance of an endocrine balance, the study of vitamin supply and the influence of light and outdoor life are subjects worthy of consideration in the problems of the restoration of a better osseous development. The etiologic factors are evolutionary and devolutionary through many generations and ages.

The shape of the head of the Caucasian varies at the present time decidedly from that of centuries ago. Anthropologists say that the relative proportion of the face and calvarium

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has undergone certain changes through the ages until now the facial parts of our race are diminutive and compressed as compared with other parts of the body. This change to artificiality is of course greatly reinforced by the relatively increased difficulty of parturition. It would seem logical to believe that prenatal as well as postnatal processes are increasing the necessity for operation on the nasal passages. It has been stated that some 80 per cent bear the mark of asymmetry or abnormality.

Under the light of modern scientific investigation many other factors are advanced in explanation of cranial malformations and deviations from normal average form.

A study of 22,000,000 school children reveals an astounding percentage of defects that may be attributed to faulty nutrition for the most part.

The biochemic problems in relation to the bones of the head that particularly interest us are met in an effort to relieve or modify asymmetry and bony abnormalities of the nose or mouth cavity, in the form of septal deflections, spurs, exostoses, enchondroses, high arch palate, crowded teeth, tonsils and adenoids.

While surgical technic has received volumes of literature, only meager consideration has been given to the underlying etiologic factors and preventive problems, the solution of which would eliminate much surgery and orthodontia.

In the field of biochemistry, as related to the cell life concerned with the development and timely calcification of bone, its growth and development, are many mysteries. Their factors as we unravel them are intimately associated with interesting studies in vitamins A, C and D particularly and an associated relationship of hormones found in the anterior pituitary lobe and thyroid.

The hypopituitary child has been studied by the Research Department of the Detroit College of Medicine and collected from 263,000 school children into special classes. The preliminary work of Drs. Kimball and Marinus classifies under this type a child who is undersized, with a small face and pinched expression, high arched palate, enlarged tonsils and adenoids, small bony structure in general, small accessory sinuses, anemia. Defective teeth, with loss of the first molar, caries,

irregular and crowded teeth, with a loss of enamel, are marked. The mother of these children may give the history of hypothyroidism, and the child may show a small adenomatous nodular thyroid. An associated mental deficiency in this type may follow in the train of a goiterous history in the mother who has had frequent pregnancy or serious illness with lowered metabolism. These children have developed an unstable nervous system and exhibit a similar but atypical variety of malnutrition. The hyperpituitary type shows the phenomena of acromegaly with abnormal overgrowth of the bones of the face, hands, and feet particularly.

According to Robertson of the University of California, two distinct substances may be isolated from the anterior lobe of the pituitary, one controlling the growth of bones and another the development of the sex organs. A case of unilateral acromegalia, under study by Kimball at the Charity Hospital, Cleveland, may give additional understanding to some of these endocrine problems as related to the skeletal structure that are at present in the realm of theory and research. Experiment, clinical findings, and the laboratory of nutrition in particular, prove that the bony affections of the head are profoundly influenced also by the supply of vitamins, especially A, C and D, and the ratio of phosphorus and calcium.

A true scientific understanding of the bony defects of the nasal passages and mouth cavity and their prevention is dependent upon the study of biochemistry of the cell as it is affected by a proper supply of vitamins and mineral salts. The lack of proper food and sunlight as a problem of preventive otolaryngology in relation to the bones of the head is especially demonstrated in the study of rickets. Although this disease was described by Glisson in 1650, it was little understood until it was produced, in 1921, by experiments on animals. The experiments of Kaufman, Crickmur and Schultz assert that in rickets in rats there occur abnormalities of the osseous capsule of the internal ear which are identical with those changes in the long bones characterized in rickets and may interfere with auditory function. Stepp and Fridenwald produced both rickets and xerophthalmia by a diet very low in vitamin A and phosphorus but rich in calcium. Nonrachitic infants and young children have between 10 and 11 mg. of

calcium and about 5 mg. of inorganic phosphorus per hundred grams of serum. All children under 2½ years with less than 3 mg. of phosphorus had active rickets. Kramer, Casparis and Howland report the increase of phosphorus with the quartz mercury light is the equivalent of cod liver oil. Orr, Hotl, Willin and Borne believe that cod liver oil and the quartz light promote absorption of calcium and phosphorus from the intestine. Neither therapeutic aid, however, meets the defects in the composition of the diet, directly or indirectly, by supplying calcium or phosphorus, but by raising the potential of cellular activity and cell selection toward the vitamins and salts that influence ossification and calcification. Goldblatt and Scrames observe with others that liver taken from irradiated rats is growth producing, whereas the livers from nonirradiated rats are inactive. The same was found true of lung and muscle tissue. It is demonstrated that experimental rickets may be produced in rats by an inadequate supply of vitamin D and disturbance of the calcium and phosphorus relation increasing with digestion. To produce rickets we must have vitamin D lacking with phosphorus low, and the calcium disproportionately high or the reverse—calcium very low and phosphorus high disproportionately. Normal calcium is 10 to 11 mg., per 100 cc., lowered in rickets to 9 or less; phosphorus, 5 to 6 mg., in rickets 2 mg. or less.

The supply of mineral salts is of great importance in addition to vitamin D. Sherman and Gillet determined that in 92 New York families examined 53.2 received a substandard allowance of calcium; 48.9 were low on phosphorus; 41.3 were below the standard of iron. To counteract this a quart of raw milk was advised for children from 3 to 13 years. In addition the growth of bones and general nutrition may be advanced by raw vegetable juice, wineglassful made from 2 carrots, 2 potatoes, 2 beets and a bunch of celery, put through a meat press, filtered, cooled and seasoned. This combination with milk has a high vitamin supply of B, which is now considered to have the Beriberi, growth, Pellagra and condition factors isolated. Chaney and Blunt have shown that calcium retention may be greatly increased by the addition of orange juice to the diet and vitamin C also may influence the calcification of bone.

A study of the geographic distribution of the disease shows it is rare in the Arctic regions and the Tropics. It is seldom noted where breast feeding is prevalent, but increases in the industrial centers with lack of sunshine. Mountain sunshine prevents it. Rats on a rachitic diet do not develop the disease in the presence of sunlight or if given food exposed to the ultraviolet ray. The food of the Esquimaux contains a plentiful supply of vitamin D. The seasonal effect is well marked by the increase of the disease during the winter months.

If the bony defects of the head are to be remedied and prevented it is necessary for the otolaryngologist to join the pediatrist and orthopedist in an application of the newer therapeutic principles that are available in this field. Rickets may be completely eradicated as successfully as on the Island of Lewis in the Hebrides. Mellanby found that the infants there were universally breast fed, and the mothers, although living in most unhygienic surroundings, partook of a diet that was in a great measure carnivorous and consisted of cod heads stuffed with cod livers, milk, fish, turnips, oatmeal and potatoes. The value of cod liver oil with the constituents of vitamins A and D, is a well established therapy, and its usefulness and scientific explanation is accepted by the laity and the medical profession. The difficulties of the administration of cod liver oil and the quantity and time interval necessary for results have lessened its usefulness. It is therefore interesting to find that biochemistry has produced a welcome substitute for vitamin D deficiency. The discovery of irradiated ergosterol as the modern antirachitic principle of great interest to the otolaryngologist as the influencing factor in bony defects of the head. It is isolated as an active and concentrated equivalent of vitamin D that can be administered in minute dosage. The preparation of ergosterol from yeast, as outlined by Greenbaum, shows it to be an impurity of cholesterol. Rosenheim and Webster found that the cholesterol was rendered antirachitic by exposure to ultraviolet rays. At the same time, independently of these investigators, Steenbock and Black, and Hess, Weinstock and Sherman made this same discovery.

Cholesterol, if highly purified by chemical means, loses its antirachitic activity when irradiated; therefore, it was evident to conclude that there must be an impurity in cholesterol which,

on irradiation, is the antirachitic principle. This impurity occurs in cholesterol only in minute quantities (1:2,000) of the cholesterol, which accounts for the fact that this discovery has so long escaped the chemists. This impurity was a sterol, which was known before under the name of ergosterol.

Ergosterol was originally isolated from ergot by Tanret and was named accordingly. It is found in yeast, in mushrooms, in many other lower plants and in certain vegetable oils. As to methods of preparation, there are various methods of extraction from ergot and from yeast. A far greater amount of ergosterol is contained in yeast, and, therefore, the methods today use yeast as a starting material. This ethereal extract contained the ergosterol, and repeated crystallization from alcohol gave crystals of a sterol, with a melting point of 154 degrees C., which seems to be identical with the ergosterol isolated by Tanret from ergot. The yeast sterol, when first isolated, melted at 135 to 136 degrees C., and many crystallizations are necessary to bring the melting point up to 154 degrees C. Solutions of yeast sterol become yellow and decompose slightly on heating or even on standing at ordinary temperature a yellow oily substance is formed.

They pointed out, as other investigators already did before them, that the reaction ergosterol—light—vitamin D, is accompanied by the appearance of a new band at 247 in the spectroscope, which in all probability is characteristic of the vitamin. Unfortunately, a very considerable concentration of pure vitamin, on the order of O. L. 1g., per 100 cc. of alcohol is necessary to secure the detection of the absorption band, so that this spectroscopic test cannot serve as a criterion for the absence or presence of the antirachitic vitamin. The authors investigated by means of this absorption band at 247, other oil for their ergosterol content and found that cottonseed oil contained from 0.75 to 2.5 per cent of ergosterol, so that cottonseed oil may be used as a new source for the preparation of ergosterol. Yeast fat contains as much as 40 per cent of ergosterol. Ground nut oil contains varying amounts of ergosterol—from zero to small amounts. Olive oil does not contain ergosterol. Linseed oil and maize oil contain small amounts of ergosterol. When ergosterol is exposed to ultraviolet light a chemical change takes place, transforming the ergosterol into

a new substance, the chemical composition of which is as yet unknown, but which, due to its absorption spectrum and its antirachitic behavior, is identical with vitamin D obtained from other sources.

In this irradiation, the source of the ultraviolet rays and the time of exposure to them are of the utmost importance. Hottinger found that the optimum time is twelve hours, and that in twelve hours of exposure an antirachitic product is obtained, of which 1/100,000,000 g. prevents the appearance of rickets in rats which are put on a ricket producing diet. This activation of ergosterol is carried out in an organic solvent, such as ethyl-alcohol or liquid paraffin or in vegetable oils.

The most interesting experiments of F. Holz show clearly that neither the exposure of ergosterol in the presence or absence of air to sunlight or artificial light, will result in the formation of vitamin D, but will produce substances chemically related to ergosterol but inactive in rickets, and only the exposure to ultraviolet rays will produce vitamin D.

A considerable amount of work has been done in the activation of ergosterol with ultraviolet rays, particularly in the University of Wisconsin. Fosbinder, Daniels, and Steenbock and Kazimierz Kon have studied the action of monochromatic light on cholesterol and have quantitatively studied the photchemical activation of sterols in the cure of rickets. They purified cholesterol by means of potassium permanganate, and the so obtained highly purified cholesterol (m. p. 147° C.) was irradiated but was entirely inactive, which proved without doubt that pure cholesterol cannot be rendered antirachitic by ultraviolet irradiation and that ergosterol is at present the only substance which is specifically activated by radiant energy. They also found that the quantity of radiant energy necessary to form an amount of vitamin D sufficient to cause deposition of calcium in the bones of a rachitic rat is from 700 to 1,000 ergs necessary for 256, 265, 280 and 293 spectroscopic lines.

Irradiated ergosterol has become the modern treatment for rickets. This discovery has explained the beneficial action of cod liver oil and of the mercury lamp treatment in rickets.

Cod liver oil contains apparently the antirachitic vitamin and the mercury arc lamp treatment produced in the human body protective substances which will prevent the formation of rickets.

The dosage of ergosterol required for cure or prevention of rickets is so minute that it can be easily given to infants as a prophylactic. Oto Rosenheim and Thomas A. Webster recommend daily doses of 1/10,000 milligrams, which will cure and prevent rickets in rats on a rachitogenic diet. A. Hottinger investigated the action of ergosterol on dogs and found it to be very active. He then used it in infants, children and adults. He recommended as a dose for infants 1 milligram a day; for babies of 2 to 3 years of age, 2 to 3 milligrams per day, and for adults 5 milligrams a day. H. Strote found that irradiated ergosterol will give beneficial effects in cases of rickets, osteomalacia and infantile tetany. He recommended doses of 1 to 2 milligrams.

The treatment of a large number of cases of rickets with irradiated ergosterol by Marfan and Dollfus Odier show when given 3 to 5 mg. a day, during the twenty days with a rest of fifteen days and the series repeated twice, that there occurs a recalcification of the end of the diaphysis of the long bones, of epiphyseal ossification and the callus of rachitic fractures when they exist. This remineralization is evident at the end of twenty days; it is often complete after the second treatment; it is more often after the third. The remodeling of the bone begins during the second treatment. The cranio-tabes and the other rachitic alterations of the skull are also modified but less quickly than the other members.

Upon the symptoms concomitant with the osseous alterations, upon the muscular hypotony, upon anemia, upon the intumescence of the lymphoid organs, the action of irradiated ergosterol is inconstant, incomplete or unappreciable. However, the general nutrition is often improved, the weight increased and the nervous irritability diminished.

In the case of tetany coincident with rickets, one may state that ergosterol has a remarkable action on it. After about eight days the spasmodic symptoms and the phenomenon of Trousseau disappears, the facial signs after fifteen or twenty days;

but the latter may reappear after cessation of treatment. In a small number of cases rickets does not appear modified by the irradiated ergosterol, without one being able to attribute it to the poor quality of the medicament, or, as one has maintained, to the fact that its action has been hindered by grippe or a concomitant febrile affection.

With the exception of several differences, the action of irradiated ergosterol is analogous to that of the direct application of ultraviolet rays. If the ergosterol appears to calcify readily, the ultraviolet rays modify more rapidly and more completely the form of the bone; the general eutrophic action of the latter is more evident. But the ergosterol has the advantage of being employed more easily and is less expensive. This medicament is then a valuable acquisition and ought to take a place of prominence in the treatment and prevention of rickets. However, more completely than does the ultraviolet rays, ergosterol when activated appears not to act upon the cause of rickets itself but upon the processes which bring about the lesions of the bone.

The application of the use of vitamin D and sunlight is of interest in the schools. As a member of the Detroit Board of Education, with the care of 265,000 children, this task of the segregation of the underprivileged child into special classes for study was adopted. The development of open air schools, special diets and treatment were provided. Our cafeteria feeds 50,000 children a day where milk, eggs and leafy vegetables are supplied abundantly. Five years ago 42 per cent had goiter; today 7 per cent exhibit this condition. We studied the problems of sunlight in the schools and furnished one room with Corex glass that was advertised to reflect 90 per cent of ultraviolet rays into the schoolroom. The glass proved worthless and developed a strain on the eyesight. In two months it was covered with a fog that resembled smoked glass. Our money was refunded and the glass removed.

## CONCLUSIONS.

1. The structure, stability and growth of the bones of the head are dependent upon the supply of vitamins A, C and D, particularly with a determined ratio of Ca and P.

2. The prevention of rickets is a prenatal problem, together with the diet of the nursing mother. The value of cod liver oil, milk, eggs and leafy vegetables needs wider application.

3. Irradiated ergosterol is equivalent to vitamin D and offers a concentrated form of preventive treatment for rickets.

4. Simple hyperplastic goiter with the attending growth problems in the Detroit public schools has diminished in five years from 42 to 7 per cent with the use of iodin.

5. Many skeletal defects in our school children, decayed and crowded teeth, small sinuses, high arched palate, bony asymmetry and deflections, congenital thyroid may be modified by prenatal care, with special attention to a supply of iodin, raw milk, egg-yolk, leafy vegetables, orange and tomato juice with irradiated ergosterol to the pregnant and nursing mother.

62 Adams Ave.

LIII.

# THE NECESSITY FOR A SCIENTIFIC INVESTIGATION OF THE CAUSES OF HYPERTROPHY OF THE LYMPHOID ELEMENTS OF THE PHARYNX.*

By D. Bryson Delavan, M. D.,

New York.

The Journal of the American Medical Association in a late issue said editorially: "A recent Government report estimates that about one-third of all operations since 1924, among the American urban population, were for the removal of tonsils or adenoids. The urgent need for comprehensive statistical studies of the results is apparent."

Among us of the American Laryngological Association the prevalence of tonsillar disease is too well recognized to need explanation or its effects to require argument. This is particularly true of the hypertrophic conditions of the lymphoid tissue of the throat.

Half a century ago the followers of Sir Morell Mackenzie, greatest of laryngologists, were diligently applying his excellent modification of the Physick tonsillotome—the first of its kind—for the removal of enlarged tonsils. His operations like those of his numerous followers, here and abroad, were attended with no abscesses of the lung or, in the main, with other untoward effect except an occasional hemorrhage, and were followed generally with brilliantly curative results. The "submerged tonsil" had not yet received full recognition.

Under his teaching, especially as related to operations upon the tonsils, and later on to the removal of adenoids under general anesthesia as first practiced by him, I pursued my own professional career with great enthusiasm, as in course of time

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did many others. Many cases sent by the best physicians were thus treated. After much experience and spurred to the idea by that brightest star of our firmament, the late Dr. John Nolan Mackenzie, when he published his clarion address entitled "The Massacre of the Tonsils," it dawned upon me that operative work upon the tonsil was becoming startlingly widespread. Meanwhile large tonsils continued to present themselves in ever increasing numbers and to be as promptly removed. My operative zeal began to wane and there arose a spirit of ever deepening mortification that nothing but surgery was being resorted to.

What could be the reasons that so many children were thus affected? Why could not these reasons be discovered and, being recognized, measures instituted to prevent the development of this so universal trouble?

Since that somewhat distant time the etiology of other diseases, many of them far less prevalent, has been and is being investigated in numerous noted laboratories, often at great intellectual and material expense. In no other period has medicine made greater advances in the realm of discovery, while countless sums of money are being devoted to problems still unsolved.

The study of the physiology and pathology of the tonsil has for many years been attempted, but often in a desultory and speculative manner, with little or no practical result excepting as shown here and there in such pioneer work as that of our friend Dr. French and a few others of his kind. What is now particularly demanded is not the study of measures for the treatment of the diseased tonsil—of that the literature is overflowing, while the socalled methods of individual operators fill the land. The real need is for the study of the prevention of the conditions which have given rise to so much abnormality and discomfort and to the consequent resort to surgery.

If it be desirable that time, money and highly superior intelligence be spent in the search for the causes of other less common diseases, it is highly important that a condition so widespread and so fraught with present difficulties and future evils should be understood and prevented.

I therefore take this opportunity earnestly to advance the proposition that at once and without further delay the lymphoid

elements of the pharynx be scientifically studied, their development, functions and pathological conditions definitely ascertained and demonstrated; and, most particularly, that accurate knowledge be secured as to the measures which should be instituted for the prevention of the conditions which exist today, to the serious detriment of the community and to the discredit of modern medicine.

I would therefore urgently recommend that the same principles be adopted for the accomplishment of this purpose as are being used in the solving of other great pathologic problems, and in particular that the investigation of the basic causes of hypertrophy of the lymphoid elements of the pharynx—hereditary, developmental, endocrine, hygienic, dietetic, climatic and otherwise already foreshadowed in our symposium of today as well as by our President in his admirable address, be undertaken by a group of highly trained research workers; that for this purpose adequate financial support be supplied, suitable laboratory facilities provided, and every possible aid given by the medical profession at large.

For the successful management of this most desirable project there should be not a mere "committee" but a central responsible body, representative of the best talent in the country, the high standing of whose members would impart dignity, intelligence and energetic effort to the work. For the fulfillment of this, no organization could exert so great an influence in bringing about the existence and the success of such a movement as could the American Laryngological Association.

I therefore commend this proposition, believing that any advance thereby gained would worthily carry on the traditions of our precedessors, and so uphold the credit of the profession and promote the welfare of the world.

LIV.

# DEAFNESS IN SYPHILIS: AN AUDIOMETRIC STUDY.*

By Dana W. Drury, M. D.,

Boston.

The following study is the outgrowth of a series of observations made in connection with a general diagnostic service in which emphasis is laid on the diseases of the ductless glands. The theory of operation of this service had been to base the initial study on a thorough physical examination, a carefully complete medical history, and a series of standardized laboratory procedures. This basic study is supplemented by additional measurements, observations or clinical studies which may be suggested by the data of the first part of the examination.[1] As may be anticipated, where a large number of cases are receiving so thorough and comprehensive a survey, individual points, in themselves of seemingly minor importance, recur with a sufficient frequency to attract attention. A correlation of the occurrence of any one of these with all of the other data of the cases presenting it may bring to light a fact of etiologic significance or diagnostic import.

In the present instance all cases presenting to the diagnostic service giving a history of a possible impairment of aural acuity, of disease of the ear, or other consideration dealing with the organ of hearing, or who in the physical examination demonstrates the actual or possible presence of any one of these, is given an audiometric study as a part of the examination. As this operation is a routine and the selection of the cases made by the examining physician, the aurist who ultimately passes on the audiometric record plays no part in the initial selection. For this reason, then, the series assumes an

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(Contributions from the Evans Memorial, No. 206.)

especial significance inasmuch as they compose a sequential series on whom ear studies have been made.

The use of the audiometer under proper conditions has been of great value in the study of these ear conditions. By means of its mechanism the measurements assume a certain degree of objectivity, as the threshold is approached from both directions and the devices controlled by the operator give real evidence of an inhibiting incooperation and an equally destructive over-willingness. Furthermore, the audiometer in its several forms plots what may be designated as the spectrum of hearing, an entity that may be regarded without too great a strain on the imagination as analogous to absorption spectrum in the field of light. A few words dealing with the method of operation may preface the development of the thesis.

## METHOD.

As was noted above, the selection of the cases rests upon a putative, possible or existent disturbance of the aural mechanism. A more representative character would be given to a statistical analysis of a series if the audiogram were a routine observation with all patients approaching the clinic. As a matter of fact, that condition is now substantially in force, but as the data of this paper in largest measure were drawn from cases selected on the earlier basis, the series as a whole loses this additional advantage.

The acuity is tested by means of the audiometers devised by the Western Electric Company. Both the 1-A and 2-A instruments are used, the former offering a wider range of frequencies and greater amplitude of intensities.

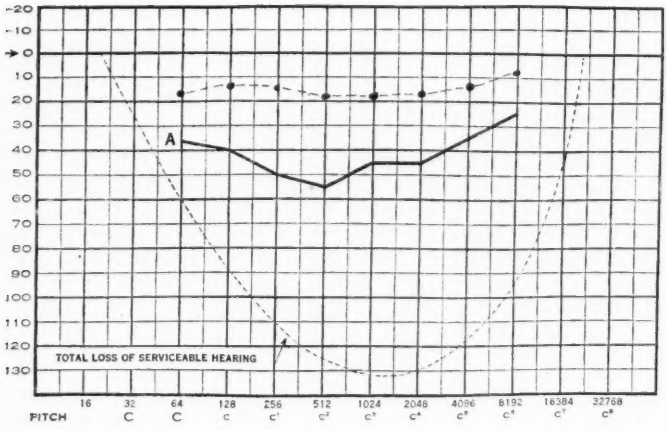
The measurements are all carried out either by the aurist himself or by a technician whom he has trained. Each point is repeated under sufficiently varying conditions so that inspection of the combined data leads to an authoritative value for the individual threshold at each intensity.

The measurements are made in a soundproof room, the construction of which has recently been described by the author.[2] This is an extremely important provision for, as the

writer has recently shown,[8] slight noises tend to lower the acuity by a very definite amount.

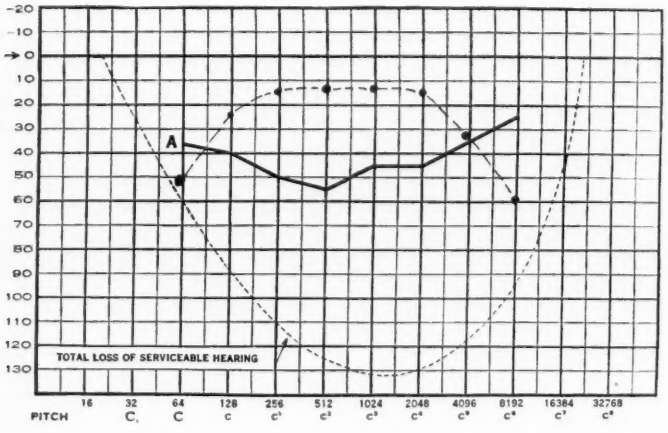
In recording the measurements, the standard form devised by Fletcher is used, and both the actual readings entered upon the graph and the percentage loss from normal hearing also determined. Broadly speaking, the shape of the audiometric curve determines six general types. To clarify the later discussion these may be illustrated.

CURVE A.

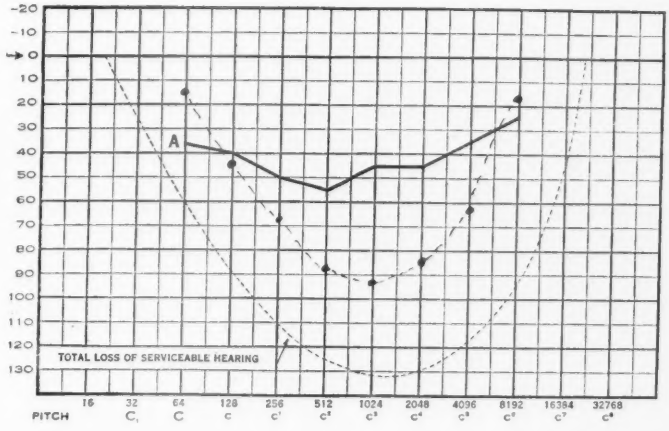



Curve A is taken from "Tests of Hearing of five hundred average cars by the audiometer No.2-A," and is a final graphic summary of the one thousand cars.

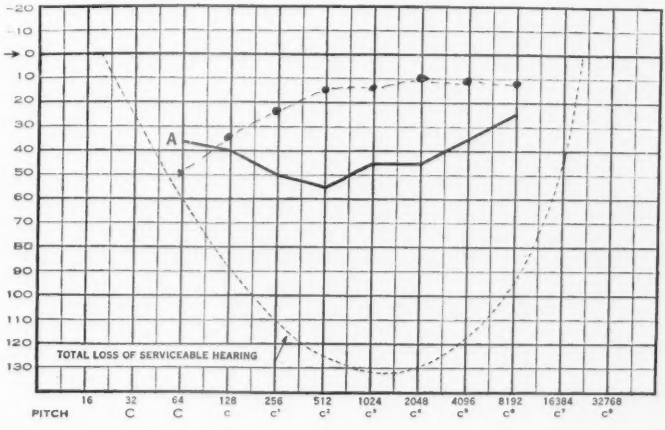
CURVE B.



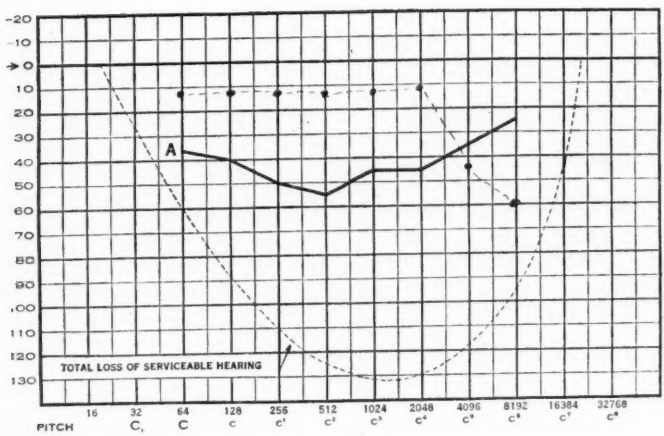
CURVE C.



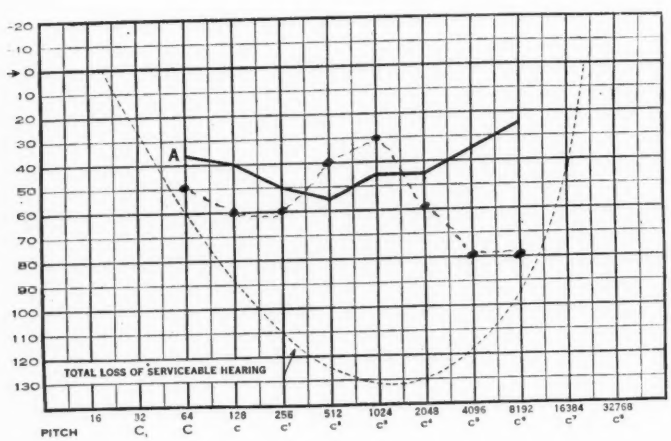

CURVE D.



CURVE E.




CURVE F.



Inspection of the graph shows that the intensities are plotted against pitches. The heavy, straight black line at the top indicates the threshold of normal hearing—e. g., the minimum intensity of sound capable of apprehension by the normal ear. The curved dotted line dependent from it represents the maximum intensities at the several pitches which can be produced by the measuring device. The curve could be projected downward by increasing the power of the instrument, but experience has shown that the intensities here indicated constitute a limit of safety and the use of larger energy might be attended by damage and injury to the aural mechanism.

From the very nature of the form of the graph it is evident that a decline at the lower and higher frequencies represents a much larger percentage loss than one of similar magnitude in the middle registers. This fact must be borne in mind in the interpretation of the shape of the curve.

*Curve "A"*—This represents a substantially uniform decrease in aural acuity, though the terminal portions, as will be noted, show a larger percentage decline than does the central portion.

*Curve "B"*—In this type of curve the principal loss is found in the high and low tones, while the speech area, depending on the position of the apex of the curve, is relatively much less affected.

*Curve "C"*—Here the trend of the loss shows the major defect in the speech area. Individuals with this type of curve are far more conscious of their hearing limitation, as the area most affected is that comprising the important sounds of every day, namely, those of the human voice.

*Curve "D"*—In this type of curve there is a selective loss of the lower tones with definitely less impairment in the middle and upper registers.

*Curve "E"*—This is the complement of the foregoing, the selective loss falling in the upper frequencies.

*Curve "F"*—Under this caption may be grouped all audiograms departing irregularly from the standard types noted above. The curve given in the plate is merely illustrative, as it is patent that an infinite variety of permutations may be grouped in this section.

The examination of a long consecutive series of audiograms showed recurrence of type "E" with a significant frequency. The present paper deals with an analysis of the cases presenting this condition, and in addition, the further experimental work which has been carried out to verify the suggestions of the initial analysis.

That the representative character of the series may be patent, a brief analysis of the total series is given in Table I.

TABLE I.

Primary Classification of Cases.

| Diagnosis | Total No. | With Audiogram No. | With Audiogram % |
|---|---|---|---|
| Endocrine: | | | |
| Pituitary | 582 | 237 | 40.4 |
| Thyroid | 364 | 126 | 33.8 |
| Gonad | 226 | 69 | 30.1 |
| Adrenal | 10 | 8 | 80.0 |
| Pancreas | 12 | 5 | 41.7 |
| Pluriglandular | 20 | 6 | 30.0 |
| Non-Endocrine | 987 | 341 | 34.5 |
| Totals | 2201 | 792 | 36.0 |

As will be seen, the total number of cases under consideration was 2,201, with a substantially equal division between the endocrine and nonendocrine group. Slightly better than one in each three cases had an audiometric study, and again, as will be noted from the table, with the exception of the small group of patients with adrenal disease, the percentage of each subgroup falls within reasonably related limits. The endocrine cases were all individuals presenting primary evidence of an unmistakable endocrinopathy. As the diagnostic study was a complete one and endocrine designation reached only after the proven elimination of nonendocrine disease which could be causal to the diagnostic picture, it is natural that many of the endocrine cases showed additional disease conditions as complications of the primary endocrinopathy. This point will be touched upon in certain of the later discussions. The nonendocrine group, as designated in the table, was a highly diversified assembly of recognized disease entities. The comprehensive character of the group may best

be ascertained by inspection of Table II, which contains classificatory designation of those conditions present in sufficient numbers to constitute a group.

TABLE II.

Etiologic Classification of Nonendocrine Group.

| Infections | Psycho-Neuroses | Cardio-Vascular Diseases |
|---|---|---|
| Arthritis | Psychoses | Cardiac |
| Tuberculosis | Neuroses | Renal |
| Focal Infections | Psycho-Neuroses | Cardio-renal |
| Toxemias | | Hypertension |
| | | Arteriosclerosis |
| **Tumors** | **Disorders of the Nervous System** | **Metabolic Disorders** |
| Malignant | Central Lesions | Obesity |
| (a) Hodgkins Disease | Parkinsonian Syndrome | Malnutrition |
| (b) Benign | Epilepsy | Allergy |
| (c) Non-Toxic Goiter | Hydrocephalus | Rachitis |
| | Encephalitis | Intoxications |
| **Blood Diseases** | Meningitis | |
| Primary anemia | Cerebral Hemorrhage | |
| Leukemias | Paralysis | Syphilis |
| Polycythemia | Mental Arrest | (A) Congenital |
| Hemophilia | Physical Arrest | (B) Neural |

Miscellaneous Disorders of the
Liver and Gall Bladder
Gastro-Intestinal Tract
Osseous System
Eyes
Ears
Skin

A study of the 792 audiograms constituting this series demonstrated the presence of 81, or practically 10 per cent, which showed a form which could be classified as of the "E" type. An analysis of the etiologic factors in these cases demonstrated a surprising incidence of putative or established syphilis. In addition there were a number of cases presenting evidences of syphilis which, however, presented audiometric observations not classifiable as the "E" type. As, however, the evidences for the presence of syphilis varied in authority, the entire group has been classified on the basis of probability and a brief summary presented in the next table (Table III).

TABLE III.

Incidence of "E" Curve.

| Syphilis | Total Cases | Cases + | o/o + |
|---|---|---|---|
| Established | 34 | 17 | 50 |
| Probable | 25 | 12 | 48 |
| Possible | 26 | 10 | 38 |
| Not Syphilis | 707 | 42 | 6 |
| With Syphilis | 39 | 48% | |
| Without Syphilis | 42 | 52% | |
| Total | 81 | | |

A word of explanation is necessary. Under "established" syphilis are listed those cases showing strongly positive Kahn and Wassermann reactions, positive spinal fluids, and those individuals whose record gives a definite history of past lues earlier established by serologic methods and clinical observation, and subject to treatment. Under the caption "probable" are listed those cases in which the serologic tests were negative, but where the examination of the eye, the neurologic studies, and various other clinical and laboratory data all pointed to the existence of a luetic taint. The "possible" group were patients presenting evidences similar in kind but not in degree to the "probable" group. Under the caption "not syphilis" are collected those cases in which a concrete nonluetic pathology was demonstrable which could account for any of the findings which might otherwise have been referred to a possible syphilitic element.

Analysis of the table shows that half of the established and probable cases gave type "E" audiograms, while in the "possible" group 38 per cent were of this character. Forty-two cases without demonstrable luetic element also showed type "E" curves, but these constituted but 6 per cent of the nonsyphilitic cases examined. In considering the probable influence of syphilis in producing this type of curve, two lines of investigation must be followed. In the first place the complicating factors of the syphilitic group must be carefully evaluated, and secondly, nonsyphilitic pathology demonstrated in the "positive" group must be analyzed in those syphilitic cases without the "E" curve. In this way, and only in this way, can the presence of a causative nonluetic factor be evalu-

ated, and equally the luetic influence properly weighed. The necessary data for the first phase of this comparison are given in Table IV.

TABLE IV.

Complications of Series with "E" Curve.

A. Endocrine.

| Syphilis | Total | Uncomplicated | Pituitary | Thyroid | Gonad |
|---|---|---|---|---|---|
| Established | 17 | 53% | 0 | 6% | 0 |
| Probable | 12 | 58% | 8% | 0 | 8% |
| Possible | 10 | 20% | 10% | 0 | 0 |
| Non-Syphilis | 42 | 0 | 31% | 24% | 5% |

B. Not Endocrine.

| Syphilis | C. N. L. | C. V. | Liver | F. I. |
|---|---|---|---|---|
| Established | 24% | 29% | 0 | 12% |
| Probable | 0 | 8% | 0 | 0 |
| Possible | 50% | 20% | 10% | 40% |
| Not Syphilis | 17% | 7% | 21% | 26% |

Over half of the "established" and "probable" cases gave evidence of no other pathology than syphilis, and even the "possible" group showed two cases in ten where a potential syphilis was the only demonstrable pathology. Turning to the glands of internal secretion as complications, we find a negligible influence in the "established" group and one scarcely more significant in the "probable" and "possible" fractions. On the other hand, 31 per cent of the nonsyphilitics had pituitary disease and 24 per cent a functional disturbance of the thyroid. The gonad would seem to play but little part, and that probably accidental, as judged by the subsequent analyses.

Turning to nonendocrine complications, one finds a significant incidence of lesions of the central nervous system and equally of cardiovascular disease in which latter series hypertension was a usual finding. The liver seemingly plays no part in this or in the "probable" group. A few cases showed focal infection. The "probable" group was singularly uncomplicated, one case showing cardiovascular disease, while the other three nonendocrine factors were unrepresented. The central nervous system, as might be anticipated, plays a more important rôle in the "possible" group, with half of the cases showing

some such condition, and a further 20 per cent of them cardiovascular disease. The liver here is not to be regarded as of marked significance, but there is a definite incidence of focal infection. In the nonsyphilitic group focal infections are the most frequently recorded complication, there is a significant incidence of hepatic disorders, and the central nervous element is not negligible. Summarized, it may be said that established or putative syphilis plays a dominant rôle, with the possibility of the pituitary and the thyroid as factors together with lesions of the central nervous system and, less certainly, cardiovascular disease, which from the frequent occurrence of hypertension may be regarded as associated with the central nervous system group. The frequency of the appearance of focal infections is regarded as reflecting the rather common occurrence of such conditions in any group of patients taken at random. Teeth, tonsils and sinuses are severally recorded in a large number of the 700 cases excluded from this discussion. While a toxemia engendered by a focal infective process might possibly be a factor, the evidence supporting this is much less than for the other entities considered.

The case for syphilis gains an added weight when one considers the group as a whole. If we select from the entire series of 81 cases with an "E" type of audiometic curve all of those which demonstrate but a single etiologic factor, the results are highly interesting. The data thus compiled are collected in Table V. Focal infections have been excluded as a factor of complication for the reasons given above.

TABLE V.

Analysis of Uncomplicated Cases with "E" Curve.

| Group | Cases in Group | Number + | o/o + |
|---|---|---|---|
| Syphilis, established | 17 | 9 | 53 |
| Syphilis, established and probable | 29 | 16 | 55 |
| Syphilis, established, probable, possible | 39 | 18 | 46 |
| Pituitary | 42 | 12 | 29 |
| Thyroid | 42 | 6 | 14 |
| Gonad | 42 | 1 | 2 |
| C. N. L. | 42 | 5 | 12 |
| C. V. | 42 | 2 | 5 |
| Liver | 42 | 6 | 14 |

Fifty-three per cent of the established cases of syphilis, as already noted, were uncomplicated. Fifty-five per cent of the combined "established" and "probable" groups, and even dilution with the "possible" fraction, offers a total incidence of "established" or putative syphilis as 46 per cent. Twenty-nine per cent of the cases showed uncomplicated pituitary disease, 14 per cent of the thyroid and liver cases respectively, while 12 per cent showed lesions of the central nervous system without other complication. Cardiovascular disease and ovarian failure drop out of consideration. That syphilis potentially plays a very important rôle in determining an "E" type of audiometric curve, must be patent from these figures, as the percentage derived from the "established" and "probable" groups together is nearly double that of the incidence of the highest nonluetic factor.

The case cannot be regarded as settled, however, until a similar comparison is made of those cases with syphilis which do not, however, show the type "E" audiogram. These data are given in Table VI.

TABLE VI.

Analysis of Cases with Possible Syphilis but Without Type "E" Audiogram.

A. Endocrine.

| Syphilis | Total | Uncomplicated | P | T | G |
|---|---|---|---|---|---|
| Established | 17 | 53% | 6% | 6% | 12% |
| Probable | 13 | 62% | 15% | 0 | 0 |
| Possible | 16 | 6% | 19% | 13% | 13% |

B. Non-Endocrine.

| Syphilis | C. N. L. | C. V. | Liver | F. I. |
|---|---|---|---|---|
| Established | 6% | 6% | 12% | 6% |
| Probable | 15% | 0 | 8% | 8% |
| Possible | 24% | 19% | 19% | 13% |

The percentage of uncomplicated cases in the "established" group is exactly that in the similar group with the positive audiogram. The endocrine element here, while slightly larger than in the positive ear group, is scarcely significant. The percentage of those patients with "probable" syphilis and a negative audiogram is substantially the same as in the group with

positive ear findings. The pituitary may play a possibly causal part here but the other endocrine entities are of less significance. One case in the "possible" group was uncomplicated with other demonstrable pathology, and there is here a higher incidence of a demonstrated endocrinopathy. Turning to the nonendocrine complications, one finds in the "established" fraction a much lower value for lesions of the central nervous system and cardiovascular disease, one somewhat lower for the focal infections, and a higher but not strikingly significant incidence of liver disease than in the type "E" group. None of the more frequent nonendocrine complications, with the possible exception of central nervous lesions, shows a significant incidence in the "probable" group, while in the "possible" fraction the incidence of central nervous lesion is but half that shown by the positive ear group, the two are identical for cardiovascular disease, there is twice the incidence for hepatic dysfunction, and but one-third for that for focal infection. In this group, as in the preceding, syphilis plays a dominant rôle, while the complicating factors are seemingly less in evidence.

Direct comparison between the positive and negative ear groups is patently impossible. In the latter there are no uncomplicated cases except those which present syphilis alone, and here the incidence is 39 per cent for the entire group as against 46 per cent for the group with positive ear findings. One would have to include the entire 700 cases which did not present syphilis to secure a group comparable to the nonsyphilitics with a type "E" curve, and this would produce a mathematical artefact devoid of any real significance.

A brief review, however, of the preceding data emphasizes the following points:

Syphilis is present more frequently than any other single factor, and, in fact, is the dominant element of the entire series. That there is a group with syphilis which does not show the characteristic ear curve is an observation which one might safely anticipate. Practically all of the cases have been treated, in the majority of instances successfully, so far as serologic tests and clinical observations could determine. The age of acquirement of the disease, the promptness of its recognition and the efficacy of the treatment must all exercise a permu-

tational influence on any end result. That syphilis is a dominant factor in producing this type of curve, the data would seem to bear unmistakable evidence.

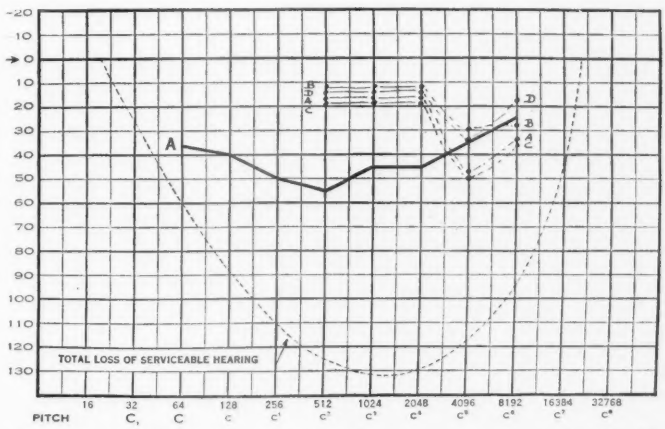
That other factors might also operate to produce a type "E" curve is the only possible deduction of the nonsyphilitic group. It is true that an ultimate possibility of earlier syphilis is very difficult to eliminate, but, in all fairness, any case that fails to give by test, history or clinical observation any evidence of a luetic infection must be adjudged as technically nonsyphilitic. A consideraiton of the complicating factors in this group as correlating with the two syphilitic factors differentiated by the ear findings is informative.

The pituitary could possibly be regarded as a factor of significance, as it appears in one-third of the nonsyphilitic cases and in practically all of these presents an uncomplicated etiology. The gross percentage of thyroid incidence is 24, the net uncomplicated only about half as much. In the two syphilitic groups approximately the same relationship obtains, and it would be a fair inference to concede the pituitary as a possible source of type "E" curve, while regarding the thyroid as constituting a much more doubtful causal agent. With its relative infrequency of appearance in all of the series, a causal influence from gonad failure may be definitely eliminated.

Turning to the nonendocrine elements, lesions of the central nervous system are patently significant. They appear as a definite factor in the nonsyphilitic group and one far from negligible in both of the syphilitic series. Cardiovascular disease shows rather wide variations, and inspection of the various values might warrant the inference that it was a significant factor only as it produced a subversive influence on the central nervous system. As previously noted, the majority of these cases presented hypertension, usually associated with arteriosclerosis. On this basis, if lesions of the central nervous system are to be considered as possibly causal agents, cardiovascular disease is but a secondary element and derives its influence through its action on the former. Focal infections as primary causal agents have already been eliminated on the basis of probability. There remain only the cases presenting hepatic dysfunction to be considered. Liver complications were absent in the "established" and "probable" groups with positive ear

findings and appear in but three cases in the same groups with negative ear findings. The incidence is slightly higher in both of the "possible" groups but hardly attains really significant proportions even here. In the analysis of uncomplicated cases, however, 14 per cent offer only hepatic dysfunction as a diagnosis. The frequent association of thyroid disease with hepatic dysfunction may be a factor in determining the relatively high incidence of both of these conditions in the nonsyphilitic group. It must be remembered, however, that in Table V both thyroid and liver disease each presented as uncomplicated factors in 14 per cent of the cases.

To summarize, then, and recognizing that longer series might modify the opinion here expressed relative to the secondary factors, it may be said that syphilis shows an incidence at least twice as large as any of the other possible factors, that the pituitary comes next in order and would seem to be also a probable causal agent of a type "E" curve, that lesions of the central nervous system and, indirectly, cardiovascular disease may exercise an influence, while thyroid failure and hepatic dysfunction are suggestive without being determining. While the case for syphilis would seem to be a well established one, control observations were felt to be essential. These have consisted of the study of untreated and of partly treated syphilis in cases without other complications. The studies with these two groups constitute the later sections of this paper.




Curve A—Syphilis and dipper curve gap..... 16—46—32
Curve B—Probable syphilis and dipper curve gap..... 12—30—27
Curve C—Possible syphilis and dipper curve gap..... 19—47—32
Curve D—Nonsyphilitic and dipper curve gap..... 13—34—18

This composite curve is the outcome of the 81 cases in Part 1 and is plotted at 2048 dv., 4096 dv. and 8192 dv. only as it graphically illustrates the gap, or we might designate it as the dipper curve.

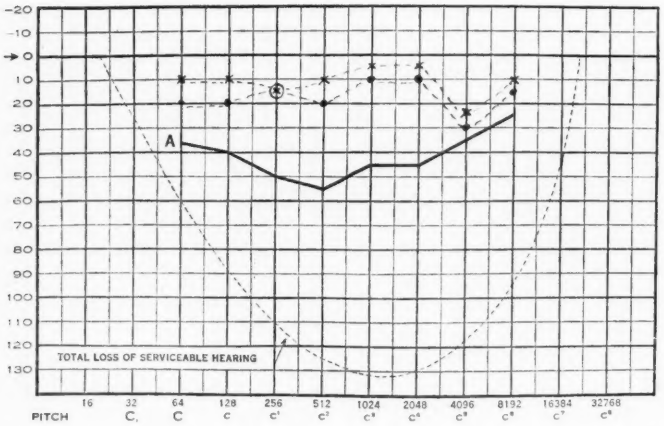
It will be noted that the drop at 4096 dv. is the greatest in the "possible" syphilitics (c) and very closely to it is the A curve of cases of established syphilis. The tendency apparently is for the syphilitic to have the lowest drop at 4096 dv.

From these 81 cases we think it fair to assume that if there is a drop at 4096 dv. of over 30 sensation units syphilis should be thought of and a closer observation made with this in mind.

GROUP B. UNTREATED SYPHILIS.

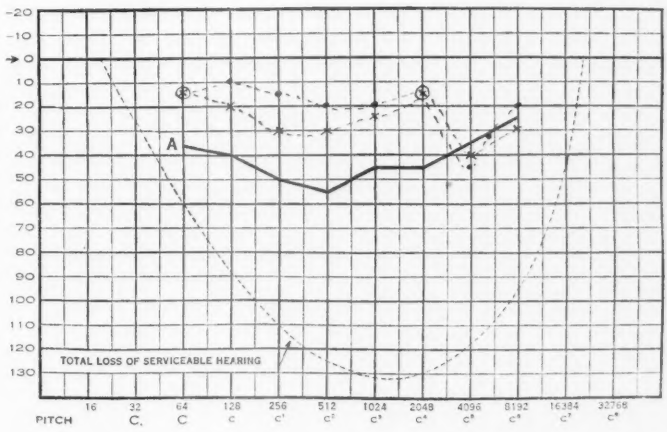
The protocols of a brief series of cases of recently acquired untreated syphilis may next be presented.

CASE I.



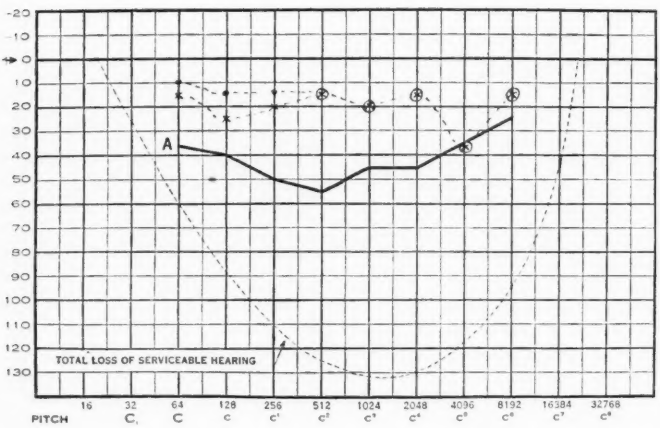

*Audiogram V-1.* Case 1.—Male, 24 years of age, was exposed to infection during February, and early in March he noticed a hard chancre on the penis. Several days following his throat was sore, intensely so, and on noting an eruption of the skin immediately came to the clinic for advice. A darkfield examination showed syphilis and many of the surface glands of the body were very easily palpable. On questioning the patient he stated that his ears were perfectly all right, he having no noise or any difficulty in hearing. The mouth showed several enlarged mucus patches and one on the left tonsil. The tuning fork test showed nothing remarkable in either ear. The characteristic audiogram shows a dip at 4096 dv., with good recovery at 8192 dv.

CASE II.



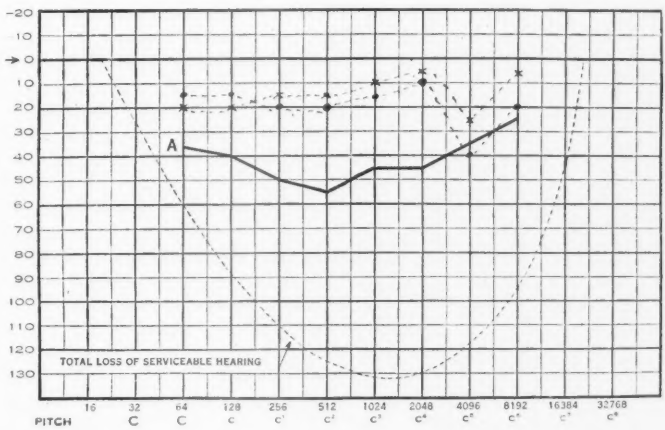

*Audiogram U-2.* Case 2.—Male, 25 years of age and white, was exposed to infection during January, with a hard chancre appearing on the penis during the middle of February and a little later a very sore and inflamed mouth and throat, together with sore glands in both groins. A beginning rash was noted over the surface of the body. A darkfield stage diagnosis disclosed syphilis, and before any treatment was instituted the ears were examined with tuning forks and the audiometer. The patient declared on questioning that his ears were quite clear, in fact, he saw no reason for them being examined. The audiogram shows a marked dip at 4096 dv. with excellent recovery at the next higher octave.

CASE III.



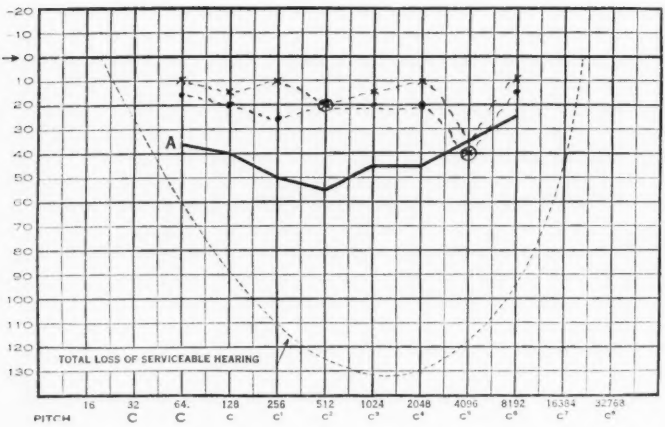

*Audiogram U-3.* Case 3.—An unmarried colored female, 21 years of age, noted a very sore mouth, which gargles used at home failed to relieve. She also noted some headache, and she thought that her hair was coming out very easily. On appearing at the clinic a primary sore was noted and a darkfield examination proved syphilis. The body glands were enlarged and several of them were very tender. The ear examination showed normal appearing drum membranes with no changes, and no changes were noted with tuning forks except she was slightly deaf to the Galton whistle. An audiogram showed a gap at 4096 dv. and recovery at 8192 dv.

CASE IV.




*Audiogram U-4.* Case 4.—A married female, 24 years of age and white, during February first noted a vaginal discharge together with an area inside the mouth which she first thought was chancre, but it failed to respond to a mouth wash and seemed to spread into the cheeks and over the tonsils. She came to the clinic for advice. On finding a hard chancre and a positive diagnosis being made by a darkfield examination I had an opportunity of examining the ears, although the patient insisted that both her ears were perfectly normal. On removing a small amount of wax from both ear canals the drums were clear and no retraction was noted. Tuning fork observations were negative. The Galton whistle showed a lowered upper level, but very slightly, indeed so slightly that ordinarily I think it would be passed over unnoticed. The audiogram showed a gap at 4096 dv. and recovery at 8192 dv.

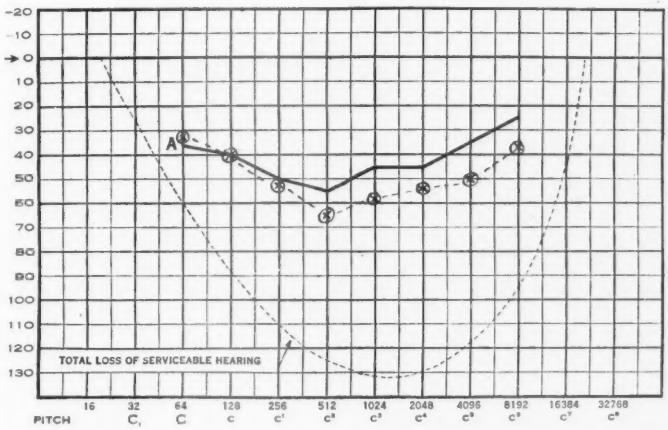
CASE V.




*Audiogram U-5.* Case 5.—A youth of 19 years was exposed about seven weeks before he noted a hard sore on the penis and enlarged glands in both groins. On the appearance of a sore throat he repaired to the clinic for advice, and a darkfield diagnosis disclosed syphilis. The patches in the mouth and throat were very pronounced and the throat was acutely sore. The ears on examination were clear and without tinnitus. In fact, the patient felt no changes in either ear. The audiogram showed a distinct gap at 4096 dv. and recovery at 8192 dv.

I had an opportunity to study these five fresh, untreated syphilitic cases through the kindness and courtesy of Dr. Austin Cheever at the Boston Dispensary. The cases were not picked but were a consecutive series taken as they appeared at the clinic for advice and treatment. Each one of the five seemed quite surprised that the ears ought to be examined, as it seemed to them purely a waste of time. In none of the five individuals was the Weber or Rinne test remarkable or notable. The middle ears of them all were also quite normal and all the tubes patent. Of course, it is conceivable that the middle ear may be affected by disease incidental to the syphilis. This situation would, of course, affect the lower range in the audiometric curve.

COMPOSITE BONE CONDUCTION (FIVE CASES), BOSTON DISPENSARY.



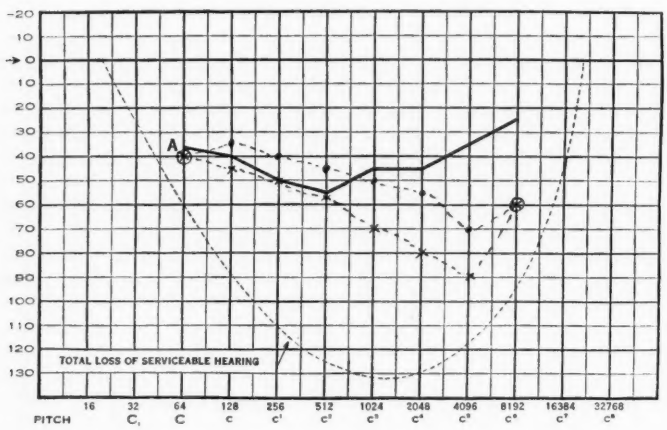

A composite bone conduction curve of the above five cases is noted below. The test was carried out with the Western Electric D-80904 bone conduction receiver. The receiver was held over the mastoid region and not by the individual being tested.

It will be noted that the figures are all below the normal bone conduction line. Bone conduction should be shorter, but with this receiver only on 128 frequency did the reading coincide with the "normal," and only at 64 frequency did it actually shorten.

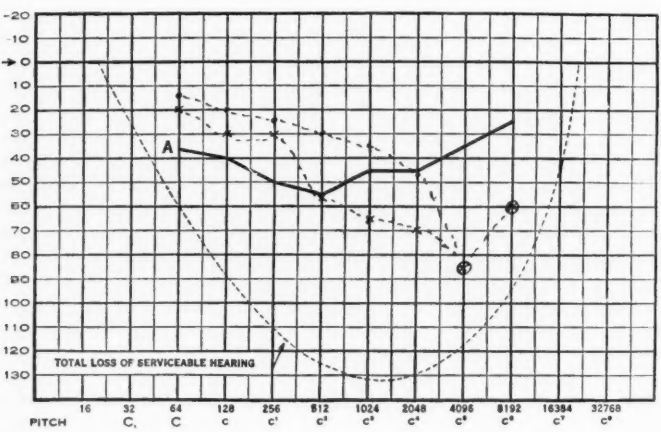
*Curve B-U.*—To the above five cases I am able to add a sixth which was sent me with the following history: A 56-year-old man, very obese but very active as a buyer for a large organization which entailed a good deal of hard work and long hours. About three months before I saw him he awakened one morning with a great deal of tinnitus in both ears and could not hear over the telephone. The deafness was very pronounced in both ears and while the low tones were affected he was unable to hear practically any of the higher notes. He noticed this when he played the piano that he was unconscious of disharmony when he struck a wrong note. He ascribed the situation to

overtiredness and to a head cold. But about the same time he noted that he was having distinct cephalic headache and his vision was failing. He then felt that perhaps a consultation would be advisable.

CASE VI (a).




CASE VI (b).



*Audiogram U-6a and U-6b.*—The ear examination showed thickened drums with very slight retraction but not enough to explain the high degree of deafness that was noticeable. While the Weber did not localize to either ear the Rinne test showed very litte bone conduction present and the Galton whistle was raised to 2.5. The tubes, while not patent, allowed air to pass into the middle ear, and it was felt that there was such a strong suggestion of syphilis that a blood Wassermann was taken, which was returned four plus. From the audiogram it is noted that a gap at 4096 dv. is very noticeable. He immediately received proper treatment by a syphilologist and a second audiogram shows some recovery at the lower end of the scale but the gap remains. Together with the antisyphilitic treatment he had several abscessed teeth removed and a generally foul mouth cleaned. This may account for the clearing of the lower acuity curve, as I believe it is fair to assume that nearly all toxemias affect the eighth nerve very definitely.

GROUP B. PARTLY TREATED SYPHILIS.

In this group I have twelve infectious syphilitic cases that I had an opportunity of studying through the courtesy of Dr. Henry D. Lloyd and the South Medical Service at the Massachusetts General Hospital. These cases were all retained in the ward while they remained infectious, which was usually a period of two weeks or more. During this time they received active medication. The study took place between the first and subsequent treatments only. They all showed active lesions at the time of the several examinations. Tuning fork tests as well as audiometric measurements, both by air and bone conduction, were done on the No. 1-A audiometer. The bone conduction apparatus used was the Western Electric's D-80904 bone conduction receiver. These twelve cases were diagnosed by darkfield methods.

That space may be conserved, the audiometic readings with this are presented in tabular form.

TABLE VII.

| Case | Ear | 64 | 128 | 256 | 512 | 1024 | 2048 | 4096 | 8192 |
|---|---|---|---|---|---|---|---|---|---|
| 1 | R | 10 | 10 | 10 | 10 | 20 | 10 | 15 | 15 |
| | L | 15 | 10 | 10 | 15 | 20 | 15 | 15 | 20 |
| 2 | R | 20 | 20 | 15 | 15 | 5 | 5 | 0 | 0 |
| | L | 30 | 35 | 30 | 20 | 20 | 10 | 10 | 15 |
| 3 | R | 0 | 0 | 10 | 10 | 0 | 0 | 10 | 10 |
| | L | 0 | 5 | 10 | 10 | 10 | 0 | 0 | 0 |
| 4 | R | 0 | 5 | 10 | 10 | 10 | 0 | 5 | 10 |
| | L | 5 | 0 | 15 | 15 | 10 | 10 | 5 | 5 |
| 5 | R | 10 | 10 | 10 | 10 | 15 | 10 | 0 | 10 |
| | L | 20 | 20 | 20 | 20 | 15 | 10 | 20 | 15 |
| 6 | R | 10 | 10 | 15 | 15 | 10 | 10 | 15 | 10 |
| | L | 10 | 10 | 15 | 15 | 15 | 15 | 15 | 10 |
| 7 | R | 55 | 55 | 30 | 50 | 65 | 45 | 70 | 60 |
| | L | 35 | 35 | 55 | 35 | 50 | 65 | 35 | 30 |
| 8 | R | 30 | 35 | 40 | 40 | 50 | 50 | 55 | 45 |
| | L | 40 | 40 | 45 | 40 | 45 | 45 | 60 | 45 |
| 9 | R | 30 | 30 | 30 | 35 | 55 | 60 | 60 | 55 |
| | L | 30 | 30 | 45 | 45 | 35 | 35 | 20 | 40 |
| 10 | R | 60 | 75 | 65 | 60 | 50 | 55 | 90 | 60 |
| | L | 10 | 10 | 10 | 10 | 10 | 10 | 20 | 15 |
| 11 | R | 10 | 15 | 20 | 15 | 5 | 10 | 20 | 20 |
| | L | 10 | 10 | 10 | 10 | 5 | 10 | 40 | 30 |
| 12 | R | 20 | 20 | 30 | 25 | 30 | 25 | 70 | 55 |
| | L | 20 | 20 | 30 | 25 | 30 | 20 | 45 | 50 |

It will be noted that the group falls into three divisions—i. e., those with no, with slight and with marked change in the audiogram. The individual case protocols follow:

Case 1 was a young man who had been exposed eight weeks before admission and when admitted had active primary and secondary lesions. The first injection received on admission following the positive diagnosis was forty-eight hours before the ear examination took place. Both canals were clean and he gave no history of ever having had any disturbance with either ear. Tuning fork tests showed nothing remarkable and the audiometric curve is quite normal. The bone conduction curve is lower at most of the points taken than is the regular points given as the usual average for the regular curve.

Case 2 was a foreigner who understood little English but said that he had never had acute hearing in the left ear since a child. He was exposed to infection in November and six

weeks following had a sore on the penis, followed by a sequence of symptoms, and came to the clinic for relief. His darkfield was positive. He immediately received his first injection. I saw his ears three days following. The left drum showed a scar of a previous infection of the middle ear, while the right drum was not remarkable. The Weber test was to the left and the Rinne was shortened in both ears. The audiometric curve together with that of bone conduction is substantially normal.

Case 3 was a young unmarried female who was infected and a positive diagnosis made by darkfield three days before I observed her, she having had her first injection on admission. Both ears were quite normal on examination, and it will be noted that the audiogram is not remarkable. The tuning forks showed no changes from a normal test.

Case 4 was a young married man of 26 years who was infected in November, the following December developed a primary lesion, and with the appearance of the secondary lesions in January appeared at the clinic the first of February for treatment, he not receiving any help during the interval, although he had taken several different medicines. I saw his ears four days after his admission and four days after his first injection, a previous diagnosis having been made with the aid of the microscope. He stated that both ears were quite normal and on inspection this was found to be the case, the tuning fork and audiometric curve both being normal.

In this same group the following eight cases make up the series of treated. Twelve cases show audiometric changes and tuning fork changes consistent with active syphilis.

Cases 5 and 6 show a slight gap at 4096 dv. in one ear with a shortened Rinne on bone conduction and an equalizing Weber test to both ears. Both individuals seemed quite surprised that the ears should be examined, as they said that they were quite normal and that they had had no tinnitus or other disturbances with their ears. In both cases the ears were found negative by inspection, the Weber test equalizing to both ears in each case and the Rinne test shortened in each of the ears. Case 5 had had a hard chancre on the penis for seven weeks, and case 6 had had secondary lesions with activity in the mouth for nearly three weeks. In both cases a darkfield diagnosis was positive syphilis.

Case 7 was rather hostile to the examination, and I felt that he was not as deaf as he claimed, although on closer questioning he denied ever having any trouble with either ear. His nose was negative, as was his nasopharynx. The audiometric curve shows depression throughout the several intensities with a slight gap in the right ear at 4096 dv.

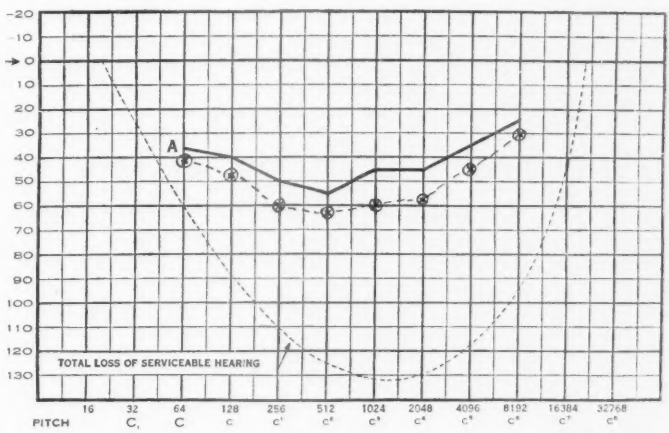
Case 8 was a widow and 40 years of age who, although denying infection, was positive by darkfield technic and had primary and secondary lesions. The course of her infection was very stormy, and although both canals were blocked with cerumen, on removal of this both drums were slightly retracted and very pale. The Weber test was localized to the right ear, and the bone conduction by the Rinne test was markedly shortened in both ears. The audiometric curve shows depression at all octaves, as well as showing a distinct gap at 4096 dv. with moderate recovery at 8192 dv.

Case 9 was a young married woman who was examined four days following her first injection. She claimed that her hearing was normal, although on inspection it was evident that she had an early dry middle ear catarrh. The Weber was localized to the left, and the Rinne test showed shortened bone conduction for both ears. The audiometric curve shows depression for the upper scale in the right ear, with depression of the left ear in the lower part of the graph.

Case 10 on examination showed an active suppurating right middle ear and complete deafness at the upper two octaves with a dip at 4096 dv. in the left ear, which on inspection was normal. The Weber test localized to the right, and the right ear showed especially a shortened bone conduction in the Rinne test.

Cases 11 and 12 both showed distinct gaps at 4096 dv. with some recovery at 8192 dv. in at least one ear. Both cases showed nothing abnormal by aural inspection, and both claimed no ear changes as the result of the active syphilitic infection.

COMPOSITE BONE CONDUCTION (TWELVE CASES), MASSACHUSETTS GENERAL HOSPITAL.




A composite curve of the bone conduction of the above 12 cases is here represented and the lengthening of the developed sound is more striking. However, none of these cases complained of their ears.

It has generally been regarded as a fact that most cases of untreated syphilitic infection are likely to develop cranial nerve lesions, and that, of the nerves involved, the eighth has more, in fact, than any of the other cranial nerves' most constantly shown early changes. It has been stated further that under the present form of treatment a larger number of cranial nerve lesions have occurred during the early stages of syphilis than by earlier records would seem to have resulted from the older methods. While this paper is not primarily concerned with treatment, I think from observation that this is not exactly the case. I offer the suggestion that possibly some of the virus may still remain potent, while in the main the general infection seems to be under control. Possibly the initial dosage is too small to do more than control the general infection. Another possibility lies in the greater care with which the earlier cases are observed, through which many matters came to light that would otherwise have been missed.

Be this as it may, I think that most observers agree that while the eighth nerve usually is the point of election, both the cochlear and vestibular branches are affected with a possibly greater frequency of the cochlear branch. These may be simultaneously affected, and it will be noted that while the vestibular branch recovers entirely, damage to the cochlear branch results in symptoms which in very many cases are permanent. In fact, so much is this the case that in later years the significant deafness will still remain recognizable. It is my opinion that the cochlear deafness does not restore, although many patients may believe such to be the case, as the principal loss of hearing in an uncomplicated case falls above the ordinary speech area. For this reason the individual is not conscious of the changes present. The tinnitus may or may not be relieved, and it becomes especially noticeable when the individual becomes overfatigued. The intensity of the tinnitus, however, may be greatly abated. On the other hand, there are individuals with untreated syphilis where the tinnitus remains even when the deafness reaches a very high degree. This is less likely to happen when the case receives proper care early in the disease. Decided variations in the hearing distance are rarely met with. Generally if improvement takes place it is gradual and not sudden. When

improvement does take place, better perception through the cranial bones also results.

While some patients do not complain of ear symptoms, I think that we have shown that the ear is involved very early by deafness even when tinnitus is not present. It has generally been assumed that tinnitus appeared first, but from the present study this would not seem to be the fact. At first, conversation is heard fairly well. This would apply, however, to early infected cases rather than to those presenting late third stage involvement. In a unilateral case of pure internal ear involvement of the milder type or where treatment is instituted early, a positive Rinne is possible, but, if the deafness increases, the character gradually changes until it becomes negative. The reason for the negative Rinne is that the bone conduction is transferred from the sound ear. Usually both ears are affected so that the positive character of the Rinne is accentuated. The fact is worthy of mention that in the nonluetic with normal hearing some reduction of bone conduction takes place, usually at about 50 years of age. On the other hand, in the hearing of the luetic with ear involvement, this shortening of the bone conduction takes place at an earlier age. Another point is also worthy of mention. In unilateral affections the Weber test is localized to the well ear, while in bilateral cases the perception of the test is entirely lost. Inflations of the eustachian tube are without effect on an uncomplicated luetic.

While it has been generally held that deafness usually occurs late in syphilis, in our observation this technically is not the case. Practically it is a very early manifestation, appearing as a gap of the dipper variety at 4096 dv. And when this situation involving both ears is found the case is greatly strengthened for a syphilitic infection. We will all agree that syphilis at once becomes an active blood infection. Now the general trend of effort in the interpretation of audiogram curves has usually been our effort to correlate their shapes and levels empirically with some pathologic condition. While certain results have been encouraging, the correlations suggest a trend rather than a defining condition.

## CONCLUSIONS.

1. That the recognition of syphilitic auditory neuritis is of paramount importance, as this may be evidence of an incipient cerebrospinal involvement.

2. That the audiometer is the instrument of choice in diagnosing early involvement of the auditory nerve.

3. That in cases demonstrating a dipper involvement at 4096 dv. in the audiometric curve, the existence of a luetic taint may be regarded as probable. Such a curve, when apprehended, offers warrant for careful further investigation of a possible syphilitic element.

4. That when a dipper gap is found in a syphilitic, the ear damage remains permanently, although the causal factor may be successfully cured.

The author desires to take this opportunity of expressing his sincerest gratitude to Dr. Allan Winter Rowe for his never failing assistance and gracious courtesies, and his thanks to Dr. Reginald Hunt for his kindness and help with the several charts.

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LV.

# FURTHER EXPERIENCE WITH THE DICHLORAMIN TREATMENT OF MASTOID WOUNDS.*

By Ralph A. Fenton, M. D.,

Portland, Ore.

The advantages of chlorin-yielding solutions in wound dressings were thoroughly discussed during the World War, and extensive literature bearing on this topic is available. Surface irritation from aqueous solvents led to the use of various oily materials which released chlorin slowly in contact with wound surfaces, and among these one of the best was dichloramin-T, 2 per cent, dissolved in eucalyptol or in chlorcosane. Obvious disadvantages in retaining watery solutions within shallow bone cavities by two-hourly injection or constant flow suggested the use of dichloramin-T to us as a first dressing in mastoid wounds, in 1918. We have continued this use for eleven years, following the details of the technic published in our report of 1921 before the Pacific Coast Oto-Ophthalmological Society on forty-five cases. At that time we reported marked reduction in duration of postoperative temperature, rapid disappearance of purulent discharge from the wound, and shortening of stay in hospital.†

Recent inquiry on the Pacific Coast shows that but few otologists who started using this method in 1921 or 1922 have continued with it. Some state that they found no advantage over a "modified blood clot." In general the discontinuance has been in the nature of a return to plain gauze packing or rubber tube drainage of the mastoid cavity, without chemical disinfection. Some surgeons stated that they disliked to bother with an additional detail at the end of such operations.

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*From the Departments of Otolaryngology and Bacteriology, University of Oregon Medical School, Portland.

Report prepared for the Pan-Pacific Surgical Congress, Honolulu, August 14-24, 1929.

†Annals of Otology, Rhinology and Laryngology, 31:214, 1922.

Careful examination of our records of several hundred cases done since our previous report bears out our earlier opinion that dichloramin-T is definitely inhibitory of bacterial growth in bone wounds. Contrary to the opinion of many, mere surgical scraping of bone is an insufficient guarantee against continuance of bacterial infestation. Macroscopically clean cavities will be found to give teeming cultures if a swab be taken just before suturing; and it is not possible to be sure that every Haversian canal is free from microbes. Of course, removal of gross lesions, necrotic trabeculæ and purulent exudates, with free washing in of uncontaminated blood and the stimulus of trauma in fostering leucocytic activity, will in most cases aid individual resistance in bringing about prompt healing without the help of bactericides.

It is, however, not infrequently a matter of tragic moment that the surgeon, especially in streptococcic cases of the type recently prevalent, can never be sure that he has reached the limit of infected bone.

For this reason we have recently conducted, with the help of the Department of Bacteriology, a careful study of the actual effect of dichloramin-T in chlorcosane upon human-blood agar plates inoculated with fresh virulent cultures from a series of acute mastoid cases, all in hospital during March, 1929. All were chosen because of the severity of their infection with hemolytic streptococci.

The examination was made as follows: Cultures from a single bacterial colony were made in infusion broth. One drop from this 24-hour culture was placed on a blood agar plate and smeared with a glass rod. Three plates were made from each culture and numbered I, II, and III. Ten to twelve drops of dichloramin-T, 2 per cent in chlorcosane, was placed on each plate II and smeared over the whole surface with a glass rod. All three plates were then incubated at 37½° C. for 24 hours. An equal amount of DCT was then placed on each plate III, smeared over the surface and incubated an additional 24 hours.

With cultures A, B, and C, 1 cc. of sterile saline solution was then pipetted on to each plate and the surface rubbed with a glass rod, to wash off the colonies. One loopful of

saline from each such plate was then inoculated into a poured blood agar plate, which was incubated 24 hours.

With cultures D, E and F, an area of about one square cm. was rubbed with a loop and subcultures made in blood agar without washing.

Cultures of F and G were made in infusion broth in three test tubes labeled I, II and III. Immediately after inoculation 1 cc. of DCT was poured into tube II. All three tubes were then incubated 24 hours. An equal amount of DCT was then poured into tube III, and the three tubes were reincubated 24 hours. Subcultures were then made by diluting in saline into blood agar plates.

Results were as follows:

| Case | Plate (no DCT) | Plate (DCT immediately) | Plate (DCT added 24 hrs. later) |
|---|---|---|---|
| | I. | II. | III. |
| A. | 23 colonies | 1 colony | 1 colony |
| B. | 1700 | 1 | 1 |
| C. | 1500 | 1 | 2 |
| D. | 2500 | 1 | 5 |
| E. | 1500 | 4 | contaminated--no strep. |
| F. | 9000 | 10 | 1 |

Broth cultures:

| | Tube I. | Tube II. | Tube III. |
|---|---|---|---|
| F. | 380 million bacteria per cc. | 840,000 per cc. | no growth |
| G. | 468 million per cc. | 11,520,000 per cc. | no growth |

Other plates were inoculated and one-half was smeared with the oil. Inhibition of bacterial growth was strikingly evident in the covered half, but on account of the red medium could not be clearly photographed. Summarizing the results of the studies by plating, which roughly reproduce the effects of bacterial growth in a film of blood-clot overlying the fresh cut bone of the mastoid, we find reduction of colonies from one-twentieth to one-twenty-five-hundredth of the number multiplying without exposure to dichloramin. In the tubes of broth, which indicate roughly the speed of bacterial multiplication in serosanguineous exudates at body temperature, dichloramin reduces growth 400 times in one case and 40 times in another during 24 hours; while after 48 hours there is no growth at

all. The average proportion of reduction of growth from the plate cultures under dichloramin is one-nine-hundredth of the ordinary amount, in this series and for these strains of hemolytic streptococci from the mastoid.

Otherwise stated, we feel that our belief is justified in the inhibitory action of dichloramin-T upon bacterial growth in acute mastoid wounds; and we feel that herein lies the explanation of the clinical facts heretofore presented, namely, the rapid disappearance of pus from wounds so treated, formation of healthy granulations, slight toxic absorption, lessened superficial necrosis and tissue loss. The resultant wound exudate is characteristically mucoid, reddish and relatively low in exfoliated cellular structures. Recurrence of yellow pus in the ear, or in a wound so treated, is suggestive of reinfection of the ear and mastoid from the throat, or of extension of the purulent process into unopened cellular structures.

Dichloramin is useful daily during the first week or ten days of the healing process; thereafter mercurochrome or silver solutions are used externally, as may seem necessary to secure smooth healing.

Our preference is still for the wide open wound, with one or two sutures at the ends. Such a wound is filled full of the oil, which is "held in" by a lightly inserted fold of narrow oil soaked gauze packing. Removal and insertion of such oil soaked material is painless: the depths of the wound are not packed, swabbed or otherwise disturbed.

Conclusion: Bacteriologic examination based on virulent cultures bears out eleven years of clinical experience in many hundred cases, demonstrating the value of dichloramin-T, 2 per cent in chlorcosane, as a routine dressing in acute mastoid surgery, not only for its remarkable inhibitory action on bacterial growth but also for shortening materially the duration of after treatment.

LVI.

# A CONTRIBUTION TO THE STUDY OF CHRONIC PROGRESSIVE DEAFNESS, WITH A PLEA FOR A NATION-WIDE INVESTIGATION.*

By Thomas Jefferson Harris, M. D.,

New York.

The most important problem today before the otologic world is the cure of progressive deafness. The last fifty years have witnessed remarkable achievements in the cure by surgical measures of acute and chronic suppurative conditions of the ear. Indeed, the history of medicine contains no fairer chapter than that having to do with the surgery of the ear. No corresponding progress has been made in the treatment of chronic nonsuppurative affections. In 1902 the writer called attention, at a meeting of this society, held in Washington, to the impasse existing at that time in the treatment of these conditions. In the intervening years, the situation has remained materially the same. During this time it is true that the pathology of that form of progressive deafness commonly known as otosclerosis has received exhaustive attention, especially on the other side of the water, but so far as a cure for it or for any form of progressive deafness is concerned it cannot be truthfully stated that we have advanced to any considerable degree. More and more it has become apparent that the cure of progressive deafness can never be secured through a knowledge of the pathology alone, and within the last few years it generally has been recognized that the all-essential need was to determine the causative factor or factors. In a number of the leading hospitals and university laboratories on both sides of the water this subject is being carefully studied. Through a grant made to it by the Carnegie Foundation, the Research Committee of the American Otological Society has begun a

*Read at the thirty-fifth annual meeting of the American Laryngological, Rhinological and Otological Society, San Francisco, Cal., July 4, 1929.

systematic and exhaustive investigation, and this committee generously has put at our disposal an audiometer for use in the work we have undertaken. For the solution of what in many ways is an almost baffling problem, as much data and information as can be obtained are highly desirable. With this thought in mind, we have carried on for the past two years a study of such material as was available. For this we have to thank Doctors John B. Rae and Arthur B. Duel for putting at our disposal the clinical material and the facilities of their clinics at the Manhattan Eye, Ear and Throat Hospital. There has been no thought on our part of presenting any new ideas. Fully as important, in our judgment, as doing this is the confirming or otherwise of investigations that have already been made.

For the satisfactory consideration of any problem, an agreement in regard to the definition of the terms employed is essential. There have been apparent differences by various writers in their understanding of the terms "otosclerosis" and "progressive deafness." The term "otosclerosis" usually has been defined on a pathologic basis. It was first employed by Toynbee to describe an ankylosis of the stapes in the oval window, and later, clinically, to denote an obstruction to the sound waves, whether of a membranous or bony nature. Politzer still later used the term to denote a primary involvement of the labyrinthine capsule, while Shambaugh,[1] in a paper read before the American Otological Society in Montreal in 1926, uses the term interchangeably with spongifying of the labyrinthine capsule, although in the same article he attempts to define it clinically, according to the location of the spongifying process, whether producing primarily a degeneration of the cochlea or a fixation of the stapes. In the first case he admits that the functional tests are indistinguishable from those of nerve deafness, while when the stapes is involved primarily the findings are those of a Bezold triad plus a normal drum and patent tube. Lucae, among others, recognizes the association of certain cases of otosclerosis with a middle ear involvement. The term "progressive deafness" also has been used with a considerable degree of uncertainty. Certain writers have classified all cases of deafness, other than those of otosclerosis, as cases of "progressive deafness." Others have included oto-

sclerosis under progressive deafness. With the attempt to overcome these objections, Sohier Bryant recently, in an article in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY,[2] has suggested the term "otitis insidiosa" to cover all forms of nonspecific progressive deafness. Kopetzky, in an article in the Archives of Otolaryngology, in 1927,[3] takes the position that progressive deafness is not a distinct entity, but that in most instances it is a local manifestation of a general metabolic disorder, and that the present classification of deafness is a hindrance to investigation and research in that it offers both a hypothesis and a conclusion. He points out that most investigators and clinicians have discarded the term "catarrhal and nerve deafness" as being unscientific, nondescriptive and misleading. He adopts for the purpose of his study the term "progressive deafness" and divides all cases into "acute and chronic." He particularly omits using the term otosclerosis. Even more radical is the position which Emerson of Boston has taken for a number of years in that he claims that all cases of progressive deafness are characterized by loss of tone perception and that the sound conduction apparatus is only incidentally and temporarily marked. This view has been vigorously assailed, but at the recent meeting of the American Otological Society, in Atlantic City, it was received with much more favor. It certainly is in line with the opinion of more and more otologists today that eustachian tube obstruction as a cause of deafness, except as an acute attack, is to be discarded. The designation "middle ear catarrh" is vague and misleading and demands revision. Even to divide cases of deafness into those of catarrhal inflammation and cases of otosclerosis without inflammation is unscientific. There is, as far as we know, no conclusive evidence to show that the type of deafness generally spoken of as otosclerosis is not of inflammatory origin. Far better, in our judgment, is it to avoid all subdivisions and to speak and think of one disease alone—i. e., chronic progressive deafness, as we propose to do in this paper.

The cases we have studied were assigned by different examiners, the one controlling symptom complained of was increasing deafness extending over a time long enough to rule out an acute condition. No case where suppuration was recog-

nized was assigned, although no attempt was made by the examiner to classify upon the appearance of the drum membrane. In all we have had the opportunity of studying 82 cases. These represent the majority of all cases of young adults complaining of progressive deafness without a history of suppuration, who presented themselves during the fall, spring and winter for three days in the week in one of the largest special hospitals of the country, during the past two years. In passing, the relatively small number of such cases is to be noted. The study of these cases had for its chief object to determine whether the opinion which in recent years has gained more and more credence, namely, that in all cases of progressive deafness there is a general contributing cause, is warrantable or not. With this purpose in view, all cases were subjected to the following examinations:

1. A complete history, including any information obtainable in regard to the onset of the trouble, previous illness and possible hereditary association.

2. A careful inspection of the middle ear, eustachian tube, nose, including the accessory sinuses, throat, tonsils and teeth.

3. Functional tests of the ears, including lower and upper limit, Rinne, Schwabach, Gelle, whispered and spoken voice, rotation (in a limited number of cases).

4. Audiogram, taken in a soundproof room.

5. Complete blood chemistry, including calcium, basal metabolism, Wassermann and Kahn.

6. X-ray of sella turcica and of nasal accessory sinuses and of teeth in all suspected cases.

7. Eye fields (limited number of cases).

8. Complete physical examination, including blood pressure, in all cases where any general physical abnormality was indicated.

9. Study of endocrine system.

10. A thorough study of every case giving any familial history to determine a possible inheritance factor.

For assistance in making these tests our thanks are due to Dr. Eggston, the head of the Laboratory of the Manhattan Eye, Ear and Throat Hospital; Dr. Frederick M. Law, of the X-ray Department; Dr. R. G. Cannaday, for his help in the making of the endocrine examinations; Dr. David D. Stowell,

for the performance of the physical examinations; Dr. George R. Hare, for the eye examinations; and Mrs. George Mills, Ph. D., for the painstaking investigation of the cases where there was a familial history present, as well as to Dr. C. Gall for his assistance in making the functional tests.

The analysis of the cases studied suggests a number of interesting observations: First, as to the frequency of heredity in the cases that presented themselves. The common impression is that in cases of progressive deafness, usually designated as otosclerosis, the hereditary element is an important and well-nigh constant factor. Fraser, in Turner's recent book on "Diseases of the Nose, Throat and Ear," states that in at least 50 per cent of cases the history of deafness in the family can be obtained. This represents the view generally held; some authorities even present it as high as 60 per cent, and one at least insisting that no case can be regarded a true case of otosclerosis unless there is a familial history. Our statistics of 8 per cent of seventy-five cases are greatly at variance with this. For this discrepancy there are three possible explanations. First, it is our observation that the history in regard to inheritance as usually taken in the clinic is faulty, inasmuch as the statement of the patient is accepted without investigation. Upon careful inquiry it is found that the deafness in the parent or relative is due to a suppurative process or developed in old age. Second, the percentage obtained from the private records of a specialist is faulty, inasmuch as he would, of necessity, receive only selected cases. Third, an error is liable to occur because of a lack of agreement in regard to the type of cases reported. In all of our cases which gave any possible hereditary history there was an exhaustive investigation of the family by a trained assistant. The result was six cases out of seventy-five, or only 8 per cent. In spite of all possible errors, it is difficult or impossible to explain this striking discrepancy with the figures of Fraser and others. Seventy-five cases are far too few to warrant drawing definite conclusions, but our findings would suggest and abundantly warrant the conclusion that heredity as a producing cause has been made too much of; certainly it is to be excluded as an invariable factor; rather it should be called a contributing factor to a greater or less degree.

Middle Ear Findings.—No attempt was made to assign cases for study upon the basis of the drum membrane appearance. The one factor was the history of advancing deafness in a young or middle aged adult, in the absence of suppuration and extending over a considerable period of time; nevertheless, because of the generally taught and accepted view that there is a characteristic otoscopic picture of progressive deafness (otosclerosis), consisting of a normal drum membrane with, in many instances, a blush over the promontory and a wide-open eustachian tube, in conjunction with functional tests showing the Bezold triad—i. e., raised lower limit, negative Rinne, and lengthened Schwabach, we have paid particular attention, in our examinations, to these several points.

Analysis of our cases gave the following results in these particulars:

M. T.—In 71 cases, or all but 11, the appearance of the M. T. was normal. In 9 there were slight catarrhal changes. In 1, pronounced; and in 1, there was an unrecognized purulent discharge.

Lower Limit.—In 63 of the 82 cases the lower limit was raised. In 6 cases it remained unaltered.

Schwabach.—In 46 cases Schwabach was lengthened. In 20 cases it was not lengthened.

Rinne.—In 51 cases Rinne was negative. B. C. > A C. In 17, Rinne was positive. A. C. > B. C. In 8, it was negative on one side and positive on the other.

Eustachian Tube.—In every case the eustachian tube was normally open. In 3 of these, there was temporary improvement after inflation.

Comment re Schwabach: In 14 of the 20 cases where B. C. was not lengthened, the deafness covered less than three years. In 3, eight years; and in 3, over ten years.

Comment re Lower Limit: The following is shown in regard to the 6 cases in which the lower limit was not raised:

Case 1.—Of two years' duration; deafness came on before marriage; history of rheumatism. Rinne positive, Schwabach normal. Right ear, much reduction in hearing; left ear, slight. Audiogram showed lowering at upper limit.

Case 2.—Brief duration; onset before marriage. Reduction in hearing slight. Infection, probably tonsil. Rinne positive. Schwabach normal. Audiogram lowered at upper limit.

Case 3.—Three years. Tonsils negative. Rinne positive. Schwabach normal.

Case 4.—One year. Audiogram reduced at lower limit. Rinne positive. Schwabach normal.

Case 5.—Three years. Audiogram reduced at upper limit. Rinne positive. Schwabach normal.

Case 6.—One year. Audiogram reduced at upper limit. Rinne positive. Schwabach normal.

Analysis according to age, etc., of the twenty cases in which Schwabach was not lengthened shows the following:

| | |
|---|---|
| 1. Age 40—A | 11. Age 25—A |
| 2. " 58—A | 12. " 55—A |
| 3. " 40—B | 13. " 41—D |
| 4. " 34—B | 14. " 15—B |
| 5. " 25—A | 15. " 23—A |
| 6. " 37—A | 16. " 37—A |
| 7. " 16—C | 17. " 45—A |
| 8. " 16—A | 18. " 23—A |
| 9. " 19—A | 19. " 43—C |
| 10. " 25—A | 20. " 48—A |

A—14: Four under 20 years old.
B— 3: Five 20 to 30 years old.
C— 2: Three 30 to 40 years old.
D— 1: Eight over 40 years old.

Note.—Large letters represent duration of deafness:

A—Under three years.
B—Three to eight years.
C—Eight to fifteen years.
D—Over fifteen years.

This analysis in the main conforms to the classical teaching in regard to the Bezold triad. The few cases (6) in which the lower limit was not raised would not negative the observation. The number in which bone conduction was not lengthened (20), as compared with 46, where it was, or nearly 33 per cent, is more difficult of explanation. A certain proportion may be ascribed to faulty testing. The work was done, however, by a trained assistant and no error sufficient to explain it could have occurred.

Rinne.—In 17 out of 51 cases the Rinne remained positive, a result corresponding closely to the Schwabach figure of 33 per cent. The only conclusion that we feel that justly can be drawn is that the Bezold triad is not an infallible test in progressive deafness. The findings as regards the raising of the lower limit, 63 out of 82 cases, are particularly interesting in their bearing on Emerson's repeated assertion that in these cases the lower limit is not raised except temporarily, when there is an acute middle ear involvement, or after a long duration of the deafness. Inquiry in regard to the duration of the disease shows that in 20 cases the duration was three years or less; in 14, from three to eight years, and in 22, fifteen years or over. This would seem to contradict Emerson's contention and, if verified by other investigators, would serve to question the position that he has taken.

Audiograms.—The functional tests were in each case supplemented by carefully recorded audiograms. For purpose of study these were divided into four classes. Class 1, where the loss in sensation units was especially at the lower end of the scale. Class 2 represents a general lowering of the scale. Class 3, a lowering at both ends. Class 4, a lowering chiefly at the upper end. To this was added a Class 5, to cover cases where there was a complete loss throughout the entire scale. The result of this classification was as follows:

| | |
|---|---|
| Class 1— 3 cases | Class 4—25 cases |
| " 2—41 " | " 5— 4 " |
| " 3— 7 " | |

The audiograms were further studied according to the duration of the disease. This study shows, for cases of three years or less duration:

| | |
|---|---|
| Class 1— 3 cases | Class 4—19 cases |
| " 2—10 " | " 5— 0 " |
| " 3— 2 " | |

For three to eight years' duration:

| | |
|---|---|
| Class 1— 3 cases | Class 4— 3 cases |
| " 2— 8 " | " 5— 3 " |
| " 3— 3 " | |

For eight to fifteen years' duration:

| | |
|---|---|
| Class 1— 0 cases | Class 4— 7 cases |
| " 2— 5 " | " 5— 1 " |
| " 3— 4 " | |

Over fifteen years' duration:

| | | | |
|---|---|---|---|
| Class 1— | 1 case | Class 4— | 5 cases |
| " 2— | 3 cases | " 5— | 2 " |
| " 3— | 4 " | | |

Comment.—The tests were carried out in a specially constructed soundproof room. The patients were carefully instructed to place the earpiece gently against the ear to avoid bone conduction. At the outset the bone conduction apparatus of the Western Electric Laboratory was employed but later abandoned because of apparent unreliability. Our experience with the 2-A audiometers is the same as that of others—that, for research work at least, it is too limited in its range to permit dispensing with the tuning fork for the lower limit and of the Galton whistle or monochord for the upper limit. Like all other instruments of precision used in testing hearing, it depends on the intelligent response of the patient to secure accurate results. In hospital patients, as a rule, such intelligence is not found to the extent it is in a private clientele. In spite of all these drawbacks, it offers an unexcelled means of charting the curve of hearing.

Analysis of the graphs in 41, or 50 per cent, of the cases shows an approximately uniform reduction for the entire range of the scale, while 25, or something less than one-third of the cases, show lowering at the upper end chiefly. Lowering at the lower end occurred but once, and reduction at the two ends, contrary to Kopetzky's experience, was only occasional.

A further study of the audiograms, based on the relation of the curve to the duration of the deafness, gave the following: In 12 out of the 27 cases, where the upper limit chiefly was involved, the deafness had its onset within three years; whereas, 13 of 24 cases (three years or less), which fell in Class 2 (reduction for entire scale) were cases of beginning deafness (three years or less), virtually the same.

This would seem to permit the conclusion, so far as the audiograms are concerned, that any dicta regarding a particular audiometric curve, based upon the duration of the disease, are impossible; further, that Emerson's contention that in

all cases of beginning deafness the upper scale limit is only or chiefly affected is open to serious question.

Focal Infections.—With Emerson and many others, we have for a long time been a believer that focal infections, especially those proceeding from the nose and throat, are an important contributing cause in progressive deafness. On that account we paid particular attention in the study of our cases to possible foci of infections. In every case an examination was made of the nose and throat. In many cases X-rays were taken of the nasal accessory sinuses. Whenever the condition warranted the case was seen by a member of the throat staff.

We recognize the possibility of overlooking focal centers in the pressure of the work, but, allowing for a reasonable percentage of error, it is hard to reconcile the results secured. Altogether 14 cases of diseased tonsils and 3 cases of sinusitis (none of the latter pronounced), were found; 2 cases of pyorrhea; 6 cases with intestinal symptoms, not extensive; 1 case of diabetes, and 6 cases, seemingly the result of an acute toxemia—quinin 1, poliomyelitis 1, typhoid 1, ptomaine poisoning 1, mumps, 1, and grippe 1.

The most careful search failed to discover more. Even in the case of the tonsil, a considerable number of cases had had their tonsils removed at the onset of their deafness, without benefit. Indeed, several cases insisted that their hearing had been made worse by the operation.

We do not attempt to explain the variance between these findings and those of Emerson. It would seem to show that while focal infection is undoubtedly a cause, too great importance has been placed upon it.

Disturbed Metabolism and Endocrine Malfunction.—In recent years these have received widespread attention in their relation etiologically to progressive deafness. The work of Rowe and Drury at the Evans Memorial in Boston is especially deserving of commendation for the painstaking and exhaustive way it has been carried on. In our cases we have sought to make a study of:

(a) Blood chemistry, including calcium.
(b) Basal metabolism rate.
(c) Urine.
(d) Wassermann and Kahn.

Many of these tests are delicate and no doubt the results show errors, but for the most part we believe that they can be relied upon, with the single exception of the basal metabolism rate. This we are convinced can lead astray, and we agree with Drury that in order to be depended upon should be repeated several times.

RESULTS.

Urine.—With the exception of a single case of diabetes, the urine was in all cases normal.

Wassermann and Kahn.—Four cases, all in women, did not show with the audiometer, nerve deafness.

Blood Chemistry.—Calcium, 34 cases above normal; 10 cases below normal. Uric acid, 37 cases above normal.

Basal Metabolism Rate.—Three cases, 3 plus; seven cases, 2 plus; thirteen cases, 1 plus.

Condition of Endocrines.—So far as possible, all cases were sent to an experienced endocrinologist for examination. We take this opportunity to express our sincere thanks to Dr. R. G. Cannaday for his time-consuming work. His reports, numbering 39, is as follows:

| | |
|---|---|
| Total number examined | 39 |
| Number with pathology | 34 |
| Number without pathology | 5 |
| Hypothyroid | 11 |
| Hyperthyroid | 11 |
| Dysthyroid | 7 |
| Hypopituitary | 7 |
| Thymic type | 8 |

It will be noticed that the majority of these are of a thyroid type, hypo or hyper. Contrary to many observers, the hypothyroid type did not prevail, neither the hypopituitary nor, as recently is being stressed, the parathyroid. It should be remarked, in attempting to draw correct conclusions of the rôle that ductless glands play in causing deafness, that in the high percentage showing gland pathology the reports repeatedly were "mild hyper or hypo," and that to the eye of the non-endocrine specialist pronounced symptoms of gland disease were not often in evidence.

Pregnancy and Childbirth.—Great importance has been placed for years by all authorities upon the rôle of pregnancy and childbirth in progressive deafness. Our statistics in this respect are of particular interest. There were 35 married women in the 82 cases examined. How many of these had children, we do not know, but probably most of the 35. In 11 the deafness had its onset after childbirth. In 8 the deafness was increased after childbirth. In 9 it came on before marriage. These figures would suggest that while not invariably a factor, pregnancy plays an important part in the progressive deafness of married women who become mothers.

Eye Fields.—Gottlieb has reported that he has found an abnormal condition of the eye fields in the cases of progressive deafness he has examined. A considerable number of our cases were thus examined but with negative findings throughout.

Blood Pressure.—Except in a few cases, the blood pressure was found normal. The total number of the cases we studied was 82. This number was materially reduced for certain tests, altogether too few to warrant or justify any attempt to draw positive conclusions.

We were handicapped in our work by being compelled to refer our cases for certain examinations outside of the hospital. Several tests which we would like to have made were omitted. The work is only a small stone in the edifice which must be erected to solve the problem of progressive deafness.

Through the single efforts of one of our members, a grant of $90,000 was made by the Carnegie Foundation for the study of otosclerosis. This grant, to cover a period of five years, was made to our fellow society, the American Otological Society. To administer this a committee was appointed consisting of Drs. Pierce, Crockett, Shambaugh, later McKernon, Duel and Wilson, all members of this society. With the money at their disposal, this committee has begun a systematic study of the problem. A central bureau, located in the New York Academy of Medicine, has been organized. Arrangements have been made with Johns Hopkins Hospital for the examination of all temporal bones sent to them and already a considerable number has been collected. Grants have been made to different individuals to finance certain special investigations,

and an exhaustive two-volume bibliography of the entire literature of progressive deafness has been compiled and within the last few weeks been put on sale. Finally, in view of the expiration of the Carnegie grant and recognizing the need of adequate funds, the committee has just completed a campaign for an endowment of $500,000, which it confidently hopes to increase in the near future to $2,000,000. The committee has given unsparingly of its time to carry on this great work. A successful solution of the problem of deafness it believes is bound to be found in due time. It is impossible, however, for this committee to do it all. The task demands the united general support of otologists throughout the length and breadth of this land.

During the past summer, Dr. Duel of this committee succeeded in enlisting our colleagues in Great Britain in the work. The active cooperation of each and every member of this society which, in its size and influence is far-reaching, is earnestly sought. A small number of centers, including Johns Hopkins, the Evans Memorial, Harvard College, are carrying on painstaking research work. This will prove invaluable.

These few centers, however, are not enough. Every city or town where there is a group or even one earnest seeker after truth can become a center in itself and assist in no uncertain terms in the solution of the problem. Every member of this society is or should be such a research worker. A few cases painstakingly studied may be of incalculable value. Complete histories, something that we busy practitioners are too often guilty of omitting, are essential. Accurate registration of functional tests, preferably with the aid of the audiometer; a thorough physical examination, including blood chemistry, and a searching inquiry into possible centers of focal infection form a part of the study of every case of progressive deafness that presents itself either in the hospital or in the private office. The securing for examination of the temporal bones of patients that have been thus studied and diagnosed is highly desirable. We all have patients who, if approached, might be willing to will their temporal bones in the interest of science. It is true that the disease has no features that would shorten life, but "otosclerotics" are as liable to perish from intercurrent infection as other people.

The problem is a big one, but never was there a time when so much encouragement existed for a successful solution. Progressive deafness does not have in it the appeal to the popular imagination which tuberculosis has, and yet, with three million school children affected to a greater or less degree, as well as millions more of the adult population, with all that it implies of handicaps to the wage earner, no greater boon can be brought to mankind than to secure the knowledge by which deafness can be not cured but prevented.

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LVII.

# A CASE OF INFECTION BY BRUCELLA MELITENSIS VAR. ABORTUS COMPLICATING TONSILLECTOMY.*

By George Fetterolf, M. D., Sc. D.,†

Philadelphia.

## INTRODUCTION.

This contribution is the story of a patient on whom I performed a tonsillectomy, and in whom four hours after the operation there occurred a chill and a rise of temperature to 105° F. Physical examination failed to reveal anything abnormal whereby the acute postoperative flare-up could be explained. However, blood studies showed that the boy was suffering from a blood stream infection by the bacillus abortus, a microorganism known since 1897 (Bang) to be the cause of infectious abortion in cattle, and for some years suspected of occurring, and in 1924 (Keefer, DeKorte, Orpen) proved to occur, in and of being pathogenic to man. Since then numerous cases have been reported from widely disseminated sources. The present one is the second to be recorded from the State of Pennsylvania.

This disease has been the subject of a broad study by Kern,‡ and the material used in this paper has been taken largely from his comprehensive study published in the American Journal of the Medical Sciences for September, 1928.

## CASE HISTORY.

The patient was a schoolboy, aged 13, with a history of good general health. His appearance was that of a youngster slightly

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‡Kern, Richard A.: The Clinical Aspects of Brucella Melitensis Var. Abortus Infection in Man. Am. Jour. Med. Sci., CLXXVI, 3, 405.

below par. He had had one attack of scarlet fever and numerous attacks of sore throat, and he presented the usual nose and throat picture found at such an age and with such a history. There had been a skin rash ten and fourteen days before I first saw him, attributed by his parents to contact with jelly fish while sea bathing. There also had been slight fever for several days, discovered by his mother, not because the boy did not feel well, but because he did not look well.

Tonsil and adenoid removal was recommended, and the boy was admitted to the University of Pennsylvania Hospital on July 5, 1928, for operation that afternoon. The usual preoperative preparation was carried out at home.

At the hour planned for the operation the boy's temperature was 100.5° F., and therefore it was decided to delay the operation for a day. The next day the temperature again was 100.5° F., and again operation was postponed. As the situation had become trying to all concerned, a medical consultation was decided on, and Dr. Richard A. Kern was requested to see the youngster and express an opinion as to whether or not operation should be proceeded with regardless of the temperature. Dr. Kern reported a negative physical examination, with the exception of a slight anemia (4,100,000 red cells; 72 per cent of hemoglobin) and a normal white cell count (7,500). He gave as his opinion that there was no contraindication to operation, and accordingly the tonsils and adenoids were removed on the afternoon of July 7th (two days after admission) under ether anesthesia.

Four hours after the operation I was called back to the hospital and found a very sick boy. He had had a five-minute chill with considerable headache, and his temperature was 105° F. Following the chill he sweat profusely. His leucocyte count was 17,000 (no differential count was made). Dr. Kern saw him that evening and again physical examination was negative.

The next day the boy was much better. His maximum temperature was 101° F., and he felt much better. His leucocyte count had dropped from 17,000 to 14,000.

Three days after the operation the leucocyte count had fallen to 5,600, with 35 per cent neutrophiles, 62 per cent lymphocytes and 3 per cent monocytes. On this same day the liver was palpable, 1 or 2 cm. below the rib margin, and re-

mained so for four days. It was not tender. The spleen at no time was palpable. A chest roentgenogram showed nothing abnormal.

The urine was normal throughout except on one occasion, when it showed a trace of albumin and a few casts.

The fever slowly declined and eight days after operation the temperature was normal. With this exception there were no symptoms of any sort after the first day.

After a week of normal temperature, fifteen days after operation, the patient left the hospital, having sustained a loss of nine pounds. The blood picture on discharge was practically the same as on admission.

Five days after leaving the hospital (twenty days after operation) the temperature rose to 102.5° F. and remained at this level for two days. There was no apparent cause and there were no attendant symptoms.

Now here comes the interesting part of the history. On the day following the operation, chill, etc., Dr. Kern elicited the fact that the boy had been drinking raw milk at a boarding school in Virginia until three weeks previously, and since that time had been using raw milk from two Pennsylvania dairies. Agglutination tests were therefore requested for the typhoid group and for brucella abortus. Dr. Frank B. Lynch of the Pepper Laboratory reported the Widal negative and the abortus test positive in 1 to 640 dilution. Another blood culture for abortus taken two days later (three days after the operation and chill) was negative at the end of ten days.

Throughout the following winter he enjoyed good health, until late in February when he had an attack diagnosed as "grip." The symptoms were those of a coryza and the temperature varied from 99 to 100 degrees F. During the latter part of April he became ill with nausea and belching but no vomiting. Again he had fever, the temperature in the evenings reaching 100 to 101.4 degrees F. After about ten days of these symptoms he was brought to the University Hospital. He presented no symptoms except a stuffy nose and a loose, deep-seated cough, the latter disappearing under appropriate treatment.

He reacted positively to B. abortus agglutination test in 1:50 dilution and negatively to 0.01 mg. of old tuberculin.

So evidently the boy is still carrying his abortus infection.

In attempting to discover the source of the infection, it was found that just before the close of school one of the school's herd of 25 cows had had an abortion, and after the close of school two others had aborted.

THE BACILLUS ABORTUS.

Now a word or two about the bacillus abortus. In 1897 Bang identified and described this organism as occurring in bovines and frequently, but not invariably, causing abortion in pregnant cows. Four years previously (in 1893) Bruce had described the organism of Malta fever under the name of micrococcus melitensis. Evans, in 1918, showed that these two, the B. melitensis and the B. abortus, are so similar as to be distinguishable only by special agglutinin absorption tests.

Transmission among the cattle occurs (a) by way of food contaminated usually by amniotic fluid or urine; (b) by coitus, the infection causing a seminal vesiculitis in bulls, and (c) in calves, by the maternal milk. The milk in 60 per cent of infected cows contains the organism, but the cattle themselves are healthy in appearance.

The question naturally arises as to how prevalent is this infection in cattle. The figures in answer to this are astonishing, but only a few will be quoted. In general it can be stated that in some regions up to 90 per cent of the herds are infected and practically no regions are free. For example, nine out of 50 herds supplying one large New York town (Ithaca) are reported as being infected, in one of the New England States (Connecticut) 90 per cent of the herds are infected, and in one of the Middle States (Pennsylvania) less than 14 per cent of the herds are known to be free of the infection. Belyea states that the infection "is widespread in the cattle in the Pacific States." Rather an appalling showing!

How pathogenic is the bacillus abortus for man? That it *is* pathogenic for man is undoubted, as the work of Evans, Carpenter and others has proved. For example, Evans produced abortion in a pregnant heifer with abortus organisms from a human case, and Carpenter caused abortion in five heifers, using five different abortus strains from human sources.

The question as to *how* pathologic for man it is, is as yet unanswered and will require much study. In 1924 the first human case was proved (Keefer) by the recovery of the bacillus abortus from the blood and urine. Since then enough cases have been recorded to justify the statement that the disease occurs more frequently than is generally known or suspected. For example, 35 have been reported from South Africa (Bevan in 1925), 23 from Italy (several observers), 35 from the United States, etc. Additional figures of a purely laboratory nature are the following:

In one series of 425 children, antibody for the abortus bacterium was found more frequently than was a positive Wassermann.

In another series, twenty cases of undiagnosed fever, five gave positive agglutination tests for abortus.

In another similar series, sixty-nine fever cases with a negative Widal, five were positive for abortus.

In another, in 783 sera there were found forty-six positive Widals and fifty-six positive abortus agglutinations.

In another, thirteen positive abortus tests were found in 1,200 sera submitted for Wassermann tests.

It is, therefore, fair to assume that many cases are being clinically overlooked, this statement being based on the fact that more than a majority of the cases on record have been discovered, not by clinicians, but by laboratory and public health workers, and by those possessing a knowledge of tropical diseases, especially Malta fever.

Now what are the symptoms of this infection? This is not the place, nor is there the time to enumerate the symptoms that have been mentioned in the various case reports, but it can be stated that they are so variable that no characteristic symptom complex can be described. The one universal symptom is fever, which may be of any type, septic, malarial, continued, etc. There is a tendency to gradual ascent, followed by gradual descent. Another notable feature is that the patients feel less ill than their fever and appearance would suggest.

The one final diagnostic test is that of agglutination.

In the last analysis very few cases will be diagnosed unless we are on the constant lookout for the disease and request

abortus agglutination tests for all patients with undiagnosed fever.

The differential diagnosis of this infection is a matter of interest. The conditions for which abortus infection have been mistaken are as follows: They will only be enumerated. The differential points in this list of diseases will quickly suggest themselves to you.

Tuberculosis—Six cases.

Subacute bacterial endocarditis—Four cases.

Typhoid fever—Three cases.

Rheumatic fever—Two cases.

Malaria—Two cases.

Pelvic inflammation—One case.

Influenza—One case.

Treatment: There is no generally successful established treatment.

Quinin apparently cured one case.

The reports on chemotherapy are a bit conflicting, but on the whole encouraging. Acriflavin was tried once, with no result. With mercurochrome some reports are favorable, some unfavorable, but this drug has proved to be the best therapeutic agent so far used.

Summary—Kern admirably summarizes the present situation, and those of his conclusions which are pertinent to the present paper are as follows:

"1. Brucella melitensis var. abortus infection is widely prevalent in cattle throughout the country.

"2. The organism is pathogenic for man.

"3. An increasing number of case reports from all parts of the country points to the growing importance of this disease as a public health problem.

"4. Especially in rural communities and small towns is the problem acute because of the twofold danger of infection: direct contact with infected animals and the commoner utilization of raw milk.

"5. There is yet a very low index of clinical suspicion of the presence of the disease on the part of general practitioners, and as a result many cases are in all likelihood going undiagnosed.

"6. Clinical consciousness of the disease will lead to the routine testing for brucella agglutinins in all undiagnosed fevers."

In conclusion I would say this: Human invasion by the B. abortus is at present, at least among otolaryngologists, so little known as to be a rarity, even a curiosity. This is because what has so well been called the "clinical consciousness" of the profession is not aroused to its prevalence. Is it not possible that we all have seen but never have observed cases of this nature? Could it not be that some of those languid, anemic children, with occasional fever, who come to us for throat and nose examination, are cases of abortus infection? Is it not conceivable that some of those children who are not cured by tonsilloadenoid surgery, by sinus treatment and surgery, combined with proper hygienic and dietetic measures, are cases of this nature? Children are the milk drinkers of the world, and could it not be that the clinical application of our knowledge of abortus infection might enable us to make a better diagnosis and apply more helpful treatment in the occasional case?

LVIII.

# THE SINUS IN PERSPECTIVE.*

By Arthur W. Proetz, M. D.,

St. Louis.

If one pause in the daily routine of his combat against sinus disease, and survey the present state of knowledge and practice of sinuology as impersonally as may be, the prospect is disquieting.

One has become so accustomed to the bewildering collection of facts, theories and superstitions of which textbooks are made, that it is difficult to reduce them to the few simple elements of which they really consist.

We know discouragingly little about sinuses, and only exert a feeble influence upon their welfare.

We know, in a word, that a sinus is an air cavity communicating with the nose; that it is capable of infection and that when its ventilation and drainage are impaired trouble ensues. We know in a general way what are the symptoms of such infections and blockades, and too often we are mistaken even in these. We have devised dozens of more or less ingenious ways of opening these cavities all for one purpose,—to let out infectious material; which sums up virtually the whole of sinus surgery, and cannot be considered a step ahead of the elementary surgical principle of draining an abscess anywhere in the body, as practiced by the ancients. Our treatment plays a pathetic rôle in the process of repair which Nature institutes and effectively pursues.

There are many exceedingly elementary matters about sinuses of which we know very little. To begin with, we do not know why they exist at all. We do not know why they are normally sterile, and why they presently become infected. We do not know why once infected they may readily recover, or why just as often they do not.

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As for their symptomatology, we know that some persons suffer intensely from sinus infections which by our present standards of observation appear insignificant.

We know, on the contrary, that some noses run copiously and continuously without putting their owners to any further inconvenience than the annoyance of blowing them.

We know that patients with symptoms which we regard definitely as arising from the sinuses may present themselves on one day in agonies of pain, and on the next in elation over its absence; and that no means at our command can detect any demonstrable difference in those same sinuses from the one occasion to the next.

We know that sometimes when sinuses are opened and neither gross nor minute pathologic changes can be demonstrated the patient is rid of his symptoms.

We know also that from a shocking percentage of sinus operations no relief is obtained; in fact, the operation often aggravates the symptoms or produces new ones. And we are still progressing by the notoriously unscientific route of trial and error to determine which cases are amenable to surgical measures and which are not.

This list of unpleasant observations is in no sense an indictment of anybody or anything. It is a plea for less action and more thought—a reminder of the inadequacy of our best methods, and a warning against the complacent acceptance of poor makeshifts for really efficient practices.

"What is wanted is not so much new data as correlation in their accumulation," contends Singer in his recent "Short History of Medicine." "The increase of medical specialism is not so much evidence of advance as it is of the heaping up of uncoordinated observations."

Naturally it must remain for the few to make the epochal discoveries upon which a science grows, but at the same time it is the responsibility of the many to unburden themselves of the charms, curses and fetishes of the past, and to familiarize themselves *in extenso* with the underlying facts of physiology and pathology. The mere mention of swollen membranes is a red rag to some of us. Swollen ankles merit only passing attention. Pus encountered in the nose is a fair indication that nature is at work successfully expelling noxious agents

from our confines, and while I do not view it as a signal for jubilation I see no occasion for hysteria in regard to it.

I am increasingly impressed with the probability that the chronicity of many sinus infections depends not upon the failure of the body to throw off some old initial infection, but upon the daily reinfection with some strain of microorganism to which the subject is nonresistant. That being the case, these patients exhibit pus in their noses at intervals in spite of anything one can do. The patient continues for months and years in a state of mild upper respiratory infection—his catarrh—which indulges in occasional exacerbations, and never quite subsides. He leaves town for a few weeks—it matters little where he goes—the trouble disappears: not a trace of it remains; he returns home and in a week's time the old sinusitis is at its old level; and this is the significant thing: *It does not recur as an acute cold which later stubbornly hangs on; but it sets in gradually and continues mild.* It is as though the patient were taking daily some noxious drug which was discontinued for a time and later resumed. To my mind this explains the sophistry "once a sinus always a sinus" which the laity now sententiously quote. Unfortunately it comes too near the truth, which is my one excuse for this diatribe.

In such cases is it justifiable to resort to treatment which experience teaches us is useless, simply to be doing something? Perhaps in occasional instances; but on those occasions certainly the something should be simple, and, above all, harmless; and not exceptionally expensive.

I spend sleepless nights thinking about the physicians of old contemplating arteries and the spurting of blood for centuries before they sensed the secret of the circulation. Fifteen hundred years elapsed before curiosity supplanted reverence for authority and amended the quaint physiology of Galen. I realize what remains to be done for sinuses, and pray that no such centuries will pass before some scientific light is thrown upon these dark cavities which lie above and beside and behind—not to say under—our very noses.

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LIX.

# CONSIDERATIONS FOR AND AGAINST CURETTING AND EXENTERATING SINUS OPERATIONS.*

By Eugene R. Lewis, M. D.,

Los Angeles.

Twenty years ago I became conscious of considerable doubt and dissatisfaction concerning much of the surgery of common inflammatory "sinus troubles." From time to time since then these doubts have been expressed in articles dealing with the common upper respiratory inflammations. The elaboration of extensive upper respiratory surgical procedures has continued in rhinologic literature and in hospital surgeries, and my contacts with postoperative results have continued; many cases drift into southern California after running the surgical gamut, hoping to restore to themselves something they have lost, vainly endeavoring to find relief from discomforts and infirmities by substituting climate for lost function. The experiences of the past nine years have served to dispel my last remaining doubts concerning radical surgical procedures. Respecting the general shortcomings of rhinologic therapeusis there may remain much to be desired; respecting radical surgery for "sinus trouble" there remains nothing desirable; it offers little or nothing of ultimate value to the patient.

First let us avoid misunderstanding terms; by "radical" surgery is meant such procedures as effect marked alteration of the normal mechanical arrangement of tissues comprising the lateral nasal wall, or of the normal cellular content of those tissues. Throughout this paper allusion will be made to radical surgery only in connection with inflammatory, infectious, allergic and other irritative processes, and nothing

*Read before the thirty-fifth annual meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, July 5th, 1929.

herein contained shall be construed as bearing upon any other abnormality.

During an acute inflammatory episode the result of almost any procedure effecting a profound alteration may appear as an improvement by contrast. The patient, suffering acutely, submits to radical operation and may soon "feel better"—but actually is not better; he is only "less uncomfortable." Temporary obstruction to drainage and nasal respiration may be almost immediately relieved, but that kind of relief is not worth while. Granting that acute suffering and nasal obstruction *may* be relieved almost immediately, neither of these reliefs are in themselves signs of real improvement, unless they are coupled with preservation of highly specialized cells and structural arrangements necessary to meet subsequent functional demands. The value of nasal surgical service cannot be measured in terms of immediate effects; the ultimate result is the only one of real value to the patient; the ambition of the rhinologist must be no less than restoration of normality. Radical surgical procedures are manifestly incompatible with such an ambition.

Treatment is not to be entered into lightly or ill-advisedly. Treatment embodies much of the art of medicine, and art is not worthy that does not touch depths of fundamentals and experiences. The goal of all treatment, health, is the composite bodily expression of satisfactory cooperations of the individual body mechanisms. Each of these physiologic mechanisms is dependent upon the operations of the others—and each is depended on by the others for much which is essential. The respiratory mechanism is of first importance as a vital organ and merits special attention to its wide ramifications. Normal sinus secretions, moved by ciliary action, emerge into the naris, where they receive dust, bacteria, pollen and other matter from the air. Through absorption areas of the upper respiratory tract and of the digestive tract these secretions effect contacts together with absorbed intestinal contents touching in upon lymph nŏdes, lymph and plasma, blood cells, endocrine and other glandular structures, hematabolist and excretory mechanisms. The resulting tissue reactions determine conditions and tendencies of individual well being or illness. Life involves a never ending succession of contacts

with adversities, entailing continuous adaptations to environment—each "adaptation" a minor ill successfully negotiated—whether physical, mental, moral, nutritional, allergenic or infectious. In early childhood is begun a series of bacterial adaptations by no means covered by the list of "contagious diseases of childhood." Immunity acquisitions are constantly taking place; most of them without symptoms, some in the guise of "febricula," of "adenoid-tonsil trouble," of "digestive disturbances," of "rashes," with or without recognizable remote manifestations such as pyelitis, myositis, endocarditis, teno-synovitis, chorea, "growing pains," "aprosexia," "night terrors," and a host of others. Usually these are destined to terminate successfully in the earlier years of the second decade. While it is important to exercise wise supervision during these critical years, with all possible consideration of systemic, nutritional, vitamin, and similar factors, it is a great mistake to seize upon upper respiratory tissues undergoing normal adaptation reactions as indications for meddlesome surgery. By the age of maturity the person has demonstrated his ability to cope with his difficulties and his individual "norm" can be fairly determined. This, taken into consideration with collateral history, physical examination and diagnostic study, will crystallize wise decisions as to what may be expected and how it may be accomplished, when a problem is confronted. These preliminary considerations are condensed in the interest of brevity; they are worthy of extensive elaboration and earnest study.

Purulent discharge from a sinus may be the expression of general systemic, metabolic, or nutritional wrong or of local tissue reaction to a foreign body, toxin or trauma, any of which may reduce local resistance sufficiently to give foothold to bacteria for otherwise impossible invasion of the tissues; with the removal of the chief etiology, the tendency is toward spontaneous restoration of the individual's normal. In a similar manner mucosal tissue reactions to allergens or vitamin deficiency may secondarily give local footholds to indigenous infections. The systemic dissemination of the typhoid bacillus, of the spirochete, of brucella, of the plasmodium, and of other systemic invaders, or of their products, frequently acts catalytically, initiating bacteriocellular reactions in nasal areas

otherwise invulnerable to infection. The same thing is true of bone fractures, shock, fatigue, toxemia, occasionally also of profound psychogenic or endocrine disturbances.

The logical major indication in meeting such conditions is to deal with the primary cause: suitable local attentions to nasal areas should not be neglected, but they should be distinctly secondary and conservative, never supplanting the measures adapted to meet the primary etiologic indications. Constitutionally inferior individuals frequently manifest, among other things, respiratory inflammations, usually of infectious type; recurring nasal evidences of bacterio-cellular conflicts are frequently diagnosed "chronic sinusitis" and these contribute considerable surgical material for radical sinus operations. Only upon the conception of the sinus inflammation having upset a previously satisfactory consitutional condition would it be logical to regard it as a real etiologic factor in these cases. If the evidence shows that a previously normal cellular and structural nasal equipment succumbed to whatever local nasal infection is conceived to justify radical operative attempts to eliminate—how much more vulnerable to subsequent similar infectious exposures is the same area bound to be, following the cellular and structural havoc left by radical operation! The clinical answer is found, written large, wherever such cases foregather.

Compare clinical observations of similar cases denied the boon of radical operation—who get on as best they may without benefit of surgery. Acute sinus suppuration tends to be self-limited. During the process, cellular and bacterial detritus become mixed with the secretions and find exit, partially or completely, depending on conditions. Ordinarily, natural processes handle the situation more or less successfully, and as it clears up quiet is restored with minimum disturbance of local conditions. The viability of the tissues is determined by the cells involved, a far safer verdict than human judgment of their prospects for recovery. Minimum alterations of cell content and structural details leave optimum local equipment with which to meet subsequent similar vicissitudes. Protective organization of exudate in and about the area is not disturbed, with the incidental dangers of increased noxious disseminations; some degree of catarrhal or respiratory trouble

may remain, the X-ray may reveal areas of cloudiness unsuspected by the patient; but he finds himself "in statu quo ante," a relatively blessed state denied the radically operated forever.

It has been observed in dogs that excision of the antral wall and removal of the mucosa is followed by complete return to normal of the tissues and structures involved. Compare this with your own observations of radical operations on human beings, or with the statements of many observers, whose findings include cicatricial basement membrane, squamous and cuboidal epithelium, vestiges of glands, general fibrotic changes, and more or less complete obliteration of the stomata and subepithelial lacunæ.

What I have to say must not be understood as a disapproval of all surgery of the nose. Conservative surgical procedures cooperating with natural defense and healing processes frequently prove of tremendous value; in critical conditions they may turn the tide, averting a fatal outcome, by timely venting of purulent accumulations. Granulomata, polyp, fibrotic adhesions, synechiæ, septal abnormalities, traumatic disturbances of normal relations and other conditions demand surgical therapy if one is to pursue the rhinologic ambition to restore normality. I am a staunch advocate of nasal surgery, provided it is conceived logically and executed conservatively. But thousands of noses have been and are still being operated upon because of nasal reactions to general allergic states; it would be as logical to excise a crop of hives and far less disastrous. There is neither time nor necessity for mentioning the numerous instances of radical operations inadvertently opening up portals of infection with resulting septicemia, osteomyelitis, sinus thrombosis, meningitis, brain abscess, metastatic pulmonitis, cellulitis and other complications far more serious than the original "sinus trouble." If these patients were playing for a possible cure it would not be quite so bad, though the odds would be pretty uneven. But they are risking serious or fatal complications with no chance of cure, even if they succeed in avoiding complications.

Radical surgery of the nasal sinuses not only fails therapeutically; it frequently is responsible for additional injury to the patient. It takes no cognizance of tissue recovera-

bility of inflamed or irritated nasal tissues, but encompasses their surgical destruction together with other essential cells not even involved by disease at the time of operation; it respects not the integrity of mechanical arrangement of nasal structures, but intentionally accomplishes profound disturbance of fundamentally essential relations of working parts of the respiratory mechanism.

From the surgical department of the University of Pennsylvania last year came the following statements: "A clinical diagnosis of chronic appendicitis implies that the patient has a localized disease of the appendix, that appendectomy is indicated, and that operation will provide a cure. That this is not the case is apparent to the most casual investigator of the results. . . . It is our well-considered opinion that patients as a whole would be better off by complete abandonment of appendectomy for so-called chronic appendicitis." I am impelled to paraphrase their statements as follows: "A clinical diagnosis of chronic sinusitis implies that the patient has a localized disease of the sinuses, that operation is indicated and will provide a cure. That this is not the case is apparent to the most casual observer of results. It is my well-considered opinion that patients as a whole would be better off by complete abandonment of radical sinus surgery."

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## LX.

# SPECIFIC PROTEIN REACTIONS IN EYE, EAR, NOSE AND THROAT.*

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Allergy is a frequent cause of respiratory symptomatology. That this is appreciated by the medical profession is evidenced by the great amount of literature now before us and the vast research that is being constantly carried on in the subject. It is our object to present a discussion of the diagnosis, symptomatology and treatment of allergy in relation to the respiratory system.

Our modern conception of allergy dates back to the work of Richet.[1] Allergy has been defined as a natural and often inherited state of human hypersensitiveness to foreign substances, usually proteins, more or less permanent in character, as opposed to anaphylaxis, an experimental condition, temporary in character, occurring only in animals. Allergic reactions may occur in almost any tissue of the body, regardless of the source or mode of entry of the foreign protein.

Predisposition to the allergic state may exist throughout life, and the symptoms appear at any age, or, if symptoms be present, disappear and the patient remain free indefinitely. We have come to the conclusion that every individual with an allergic constitution or background is potentially liable to allergic disease of some type. So long as his tolerance is high he remains free of symptoms. Vaughan[2] has termed this the "balanced allergic state." Under certain circumstances, such as in acute infectious disease, change of environment or occupation, or any condition lowering the usual state of well-being, the threshold of tolerance is lowered, and contact with the substance to which he is sensitive results in some clinical manifestation of allergy. It is a well established and accepted fact

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that such manifestations make up certain familiar clinical syndromes in the respiratory tract. These may be classified as follows:

a. Vasomotor rhinitis or Allergic rhinitis } seasonal pollenosis } hay fever
perennial }

b. Allergic bronchitis } seasonal
c. Bronchial asthma } perennial

These clinical syndromes are made up by some combinations of the symptoms classified and enumerated herewith:

1. Ocular.—Itching and smarting of the lids, injection and edema of the conjunctivæ, occasionally a granular, dry, injected conjunctiva, injection of the sclera and lacrimation.

2. Nasal.—Sneezing, particularly in the morning on arising, with rhinorrhea productive of a watery or clear mucus secretion, obstruction with resultant mouth breathing, and in childhood the facial deformity resulting from this type of breathing.

3. Pharyngeal and Oral.—Itching of the palate and ears, swelling of the uvula and soft palate and extreme dryness in mouth.

4 Laryngeal.—Cough (dry, hacking), hoarseness and not infrequently swelling of larynx and glottis, oftentimes requiring immediate treatment for relief.

5. Bronchial.—Cough, which may or may not be productive of clear or mucopurulent material. Dysnea, usually of the expiratory type, associated with wheezing, usually worse at night, but which may occur any time. Symptoms may be acute or chronic.

All these symptoms are explainable by the characteristic reaction of the mucous membrane of the allergic individual who is exposed to the protein or proteins to which he is sensitive. They occur regardless of the source or type of protein or whether the protein reaches the individual by direct contact, ingestion or absorption. There is very marked redness of the pharynx, marked hyperplasia of the lymphoid tissue, both medially and laterally, and considerable secretion of clear mucus. In the epipharynx the picture is the same. In the nose the turbinates are swollen, often to the point of obstructing drainage and nasal breathing. The mucous membrane may

be a dull red or may be pale and boggy and exuding a clear watery or mucous secretion. In old cases polypoid degeneration is common. This same vasomotor congestion may extend up the eustachian tubes, invading the middle ear and giving the drums a dusky appearance, which cannot be differentiated from the tubal or middle ear congestion resembling that found in a low grade bacterial infection. The bronchial mucous membrane, as viewed through the bronchoscope during an attack, reveals narrowing of the bronchi from spasm of the bronchial musculature, marked hyperemia with edema of the mucous membrane and mucous secretion which may form plugs resulting in obstruction.

Characteristically, then, we may expect in any portion of the respiratory tract a mucous membrane lining in which the lymphoid tissue is hyperplastic, the mucous glands hypersecreting and the tissue as a whole edematous.

Tobey's[4] description of the respiratory mucous membrane in allergic disease is quite similar to our own except that he divides them into two classes. In the first there is a simple turgescence of the nasal cavernous tissue and an increased secretion of mucus with a reddened mucous membrane. This type resembles acute rhinitis from infection. The second type is an aggravated form of the first, but with marked pallor of the entire mucous membrane, replacing the redness. Edema is a marked characteristic that places this group in the allergic type.

Proper diagnosis of respiratory allergy is based upon the family history, clinical history, physical examination, including that of the nose and throat, cutaneous testing, X-ray studies of the lungs and sinuses, and laboratory studies.

A positive hereditary history is obtainable in upward of 50 per cent of all cases of allergic disease. Some writers claim as high as 75 per cent, but in our observations in several thousand cases we have not been able to obtain so great a percentage. One should ascertain the occurrence of asthma, hay fever, eczema, urticaria, etc., in any relative of the patient. It is interesting to note and has been observed by us and others[5] that cases with bilateral family history usually manifest clinical symptoms of allergy in the first decade of life; those with unilateral family history manifest symptoms somewhat

later in comparison. However, our experience is that one with an allergic background or constitution may develop symptoms at any period of life but rarely after the fifth decade. Therefore, it is quite apparent that heredity is an important factor in determining the possibility of developing some form of allergy during life.

Personal history: Age of onset is important to the extent that as age increases the frequency of development of allergic symptoms decreases. It is rare to observe the onset of allergic disease past the age of 50 years. However, in many patients of advanced age or of the age beyond which one would expect the development of allergic disease of the respiratory tract an early history of eczema or other manifestation of allergy exists. Other important points to be brought out in the personal history are with regard to the seasonal character of the illness and the effect of change of residence. Of the frequency of so-called head colds, of periodic attacks of bronchitis of nonasthmatic character, with or without fever, and of repeated operations on nose and throat.[5] Of the character of the bedding, furniture, rugs and cosmetics used, of the effect noted from the ingestion of certain foods and of contact with certain animals or objects, of occupation and its relation to the illness. All of these are important, as any one of them may furnish an invaluable lead that may mean a correct diagnosis and proper treatment to the benefit of both patient and practitioner.

Physical examination: A careful general physical examination is equally as important in allergy as in any other medical condition. The physical findings in respiratory allergic disease are too familiar to warrant discussion further than to emphasize the necessity of differentiating them from other conditions that might simulate them. On the other hand, completely negative findings are not at all uncommon during intervals when free of symptoms. It is under the latter circumstances, and particularly when we speak of the symptoms as being doubtful in character, that a careful examination of the nose, throat and accessory sinuses by a competent specialist is an absolute prerequisite. Characteristically (Montgomery[6]) he will find a greater or lesser degree of engorgement and lymph-adenoid hypertrophy with their usual sequelæ, namely, polypoid formation and enlargement of turbinates, tonsils and

adenoids, thus explaining why many allergic individuals exhibit the signs and symptoms characteristic of sinusitis, otitis media and nasal obstruction manifested by adenoid formation.

X-ray examination of accessory sinuses is useful chiefly to rule out sinus disease, although infection, as we say, may be present coincidentally with allergic disease. The need for X-ray examination of the chest is evident in differential diagnosis.

We now come to the final and perhaps the most important examination of the allergic individual, the protein skin tests, on which we must rely in obtaining the knowledge wherewith to administer specific treatment. Since the technic of skin testing is so well known it seems needless to enter into a discussion as to the various technics or their particular merits, except to say in passing that regardless of the method employed excellent results will be obtained, providing an active protein is used and the patient is a sensitive one. The interpretation of reactions is important and we strongly feel that skill can only be acquired through repeated observation and experience. In evaluating the importance of reactions one must remember that the size of the reaction is not a measure of its etiologic significance and that the reaction obtained must be definitely correlative with the history of the patient.

Etiology.—Etiology of respiratory allergy is best discussed by study of the following classification:

Demonstrably Sensitive:

Seasonal..... { pollens / food

Perennial..... { pollens, foods, epidermals, dusts, miscellaneous, bacterial ?

Not Demonstrably Sensitive

Seasonal and Perennial

Bacterial

Physical agents (heat and cold)

Psychic

Drugs

Pollen allergy usually manifests itself as of a definitely seasonal type, divided for clinical purposes into spring, summer or fall, or combinations of all. In tropical or subtropical climates perennial pollen allergy is not uncommon.

Food allergy is the most common form of perennial respiratory allergy, although occasionally foods may cause seasonal symptoms. This type of sensitization is most common in children, and here is found the group of so-called chronic hay fever. By this term is meant a perennial nasal obstruction, constant coryza, frequent sneezing, particularly upon arising in the morning, and usually a history of numerous operations on the nose and throat, the most common being repeated adenoidectomy. Here the fact is that the repeated adenoid hypertrophy is the result of allergic disease rather than the cause of symptoms.

Epidermal or animal emanation allergy is caused chiefly by danders that cling to the hair or feathers of domestic animals, such as horse, dog, cat, and goose, chicken, duck, etc.

Under the heading of miscellaneous agents producing allergic disease of the respiratory tract, perhaps the most important is orris root, the base of most perfumed cosmetics, tooth paste, etc.

Bacteria as etiologic factors in producing allergic disease have long been under suspicion (Walker[7]). However, no one has yet demonstrated beyond a doubt that they may be considered allergic agents of the type of other allergens, such as foods, pollens, etc. It is our opinion and belief that bacterial infection, focal or general in itself, is not the etiologic factor that produces respiratory allergic manifestations. We are in accord with other observers (Todd,[8] Selfridge[9]) in the belief that infection may be present in the respiratory tract in the presence of an allergic condition, but that it is purely coincidental or secondary to the already existent allergic disease on the basis that chronic or repeated acute edema of the respiratory mucous membrane causes a hyperplasia of the lining mucosa of the sinuses, interferes with their blood supply and drainage, and causes them to become fertile fields for bacterial invasion. To repeat, we believe that the sinusitis is the result of allergic disease, but never the cause. Others, however, are not in

accord with our opinion. Walker[10] and Rackeman,[11] also Goodale,[12] believe where no reactions are obtained by skin testing that the individual is sensitive to bacteria. Gottlieb[13] believes that accessory sinus infection may cause allergic asthma and that the nonallergic type is often due to the direct irritation caused by contact of the infected secretion of the nose with the tracheal and bronchial mucosa. Coates[14] thinks that bacterial proteins elaborated on the upper respiratory mucosa must at times be the cause of sensitization of this tissue, causing allergic manifestations. Wilmer[15] classes all cases that fail to respond to cutaneous protein tests as of the bacterial type, but does not test with bacterial proteins because of the unsatisfactory, unreliable and misleading results. We are wholeheartedly in accord with this latter statement, as our experience over a 13-year period has been quite similar. We have tried to extract bacterial proteins by many methods that have been described and have used commercial bacterial proteins with only most discouraging and unsatisfactory results. Hurvitz[16] thinks that it is doubtful that an asthmatic patient can become sensitive to bacteria with which he is infected, because the largest number of observers have failed to get positive skin reactions with bacterial extracts. We, too, are in accord with most observers that secondary infection of a congested mucous membrane may serve to bring on and perpetuate certain respiratory symptoms that have an allergic basis for their existence.

It has been our experience in observing the so-called bacterial type of allergic individual that he has a history of acute infectious disease such as influenza, pneumonia, pulmonary abscess or unresolved pneumonia, from which time he dates cough, at first nonproductive, later productive and growing progressively worse, nasopharyngeal symptoms, and finally wheezing and dyspnea. This condition rarely occurs in the young individual, but usually in those past the fourth decade of life. Two explanations are here offered for this sequence of events. The first harks back to the loss of tolerance or upsetting of the balanced allergic state, of which we spoke earlier and is most common in younger individuals. The second has to do with the mechanism of production of the symptoms commonly associated with allergy. Allergy may

be regarded as merely one trigger which may set off a characteristic train of symptoms. Some mechanism other than that of allergy may do the same. For example, an injection of histamin may produce asthma. There is nothing allergic about this. The analogy between nonallergic asthma and the asthma following acute infection is evident.

Ramirez[17] reports a series of cases studied and treated endoscopically because of bronchial asthma or associated bronchitis, of which five were definitely allergic and twenty nonallergic. Of the former group, 3 were past 40, while in the latter all but six were past 40 years of age. It was interesting to note, too, that in every one of the twenty-five cases studied he was able to isolate some organism. When this condition occurs in the individual under 40 years he found that it usually followed influenza or bronchopneumonia.

House Dusts.—House dust was advanced as a cause of respiratory allergy by Cooke[18] in 1922. This investigator recognized the possibility of dust containing proteins from various and sundry sources, such as dogs, cats, feathers, rugs, etc., in the house, but claims that such is not always the case; that there is some specific substance of unknown origin in the house dust. Our experience has been that the specific substance in house dust can frequently be traced to some definite source, thus raising the presumption that in those instances in which this has not been possible it has been due to an insufficiently exhaustive search. A word, in passing, to condemn the nonspecific use of commercial extracts of dust, for it seems hardly conceivable that the dust of one city would or could be the cause of symptoms in another city, perhaps thousands of miles away.

Duke[19] has described and we are all familiar with the production of symptoms of respiratory allergy by such physical agents as heat and cold. Duke, for the want of a better name, has termed this phenomenon physical allergy. It is our opinion that a physical agent may be the precipitating factor yet scarcely the etiologic agent in the production of symptoms. In the majority of cases it is possible to demonstrate a sensitization to foreign proteins. In those in which this is not possible we should hardly say that there is a "sensitization"

to heat or cold, reserving the term sensitization for use in its usual immunological sense.

Treatment.—It is our object in discussing treatment of respiratory allergy to discuss freely specific therapy based upon skin tests, also surgical and vaccine therapy in the bacterial types, and, in passing, the symptomatic and general medical treatment.

Specific treatment is based upon the supposition that any particular protein which has given a positive reaction is etiologically important, providing that with it we can by inhalation, ingestion or by subcutaneous injection reproduce the symptoms of allergy at will, and that it is or has been present in the environment or diet of the individual.[20] To reproduce symptoms is not difficult; it is, however, dangerous and as a result seldom resorted to. On the other hand, to demonstrate the offending proteins in the diet or environment is of the utmost importance. Particularly is this true of pollen proteins, for to treat a patient with pollen protein not present in the environment, even though positively reacting, is useless and reprehensible.

Pollen.—The necessity for determining the pollen environment of the pollen sensitive individual has led to numerous extensive botanical surveys[22] of wind-pollinated plants. The knowledge so obtained correlated with the results of skin testing is the first step in successful treatment. This together with a history of the seasonal character of symptoms determines which pollens are to be used in the preparation of the antigen which is to be administered to the patient.

Preseasonal treatment is the method of choice and should be commenced about fourteen weeks prior to the anticipated season, given at intervals of five to seven days, dependent upon the time the patient appears for treatment and the interval remaining before the anticipated onset of symptoms. Coseasonal treatment is advisable and sometimes necessary when patients appear too late for the former procedure. Our method of administration of treatment is a modification of that described by other workers.

With any method, local and constitutional reactions must be guarded against. Local reactions consist of the production

of redness, swelling and intense itching at the site of injection. General or constitutional reactions consist of the production of or the aggravation of the symptoms for which the patient is being treated or a generalized urticaria. Reactions usually occur within a few minutes to twenty-four hours after the injection. Such reactions require a repetition or diminution in size of dosage when the next treatment is given. Any or all of these reactions may be controlled by the use of epinephrin chloride, 1 1000, given subcutaneously in dose 0.5 cc. to 1 cc. The result of treatment with pollen antigen in pollen sensitive individuals is very satisfactory, there being some degree of relief in more than 90 per cent of the cases.

Foods.—Specific treatment of food sensitive patients is unsatisfactory and unnecessary in the majority of cases, since satisfactory results can be obtained by elimination of the offending food proteins from the individual's diet. When such necessary articles of diet as eggs, wheat and milk are the offending substances, their elimination is a serious matter, requiring considerable ingenuity in the preparation of a satisfactory diet. For instance, sensitization to eggs means the elimination of every possible food or combination of foods that contains egg. This also holds true for other common foods. Where substitution of foods is possible it should be carried out. Specific treatment against food hypersensitiveness has been attempted but without success. Several authors have attempted desensitization by giving gradually ascending doses of the protein by mouth until such time as the individual was able to tolerate large amounts. The second method is that of giving gradually ascending doses of the protein subcutaneously. Our experience with the above described methods of desensitization has proven both unsafe and unsatisfactory. Therefore, we have advocated the elimination of the offending food substances from the diet over a period of years, depending upon the age of the patient and the results of semiannual or annual retesting. Children, as a rule, lose their sensitivity to foods as they grow older, and elimination during the sensitive period is the method of choice.

Epidermals.—The individuals sensitive to the fine epithelial scales from such animals as horse, dog, cat, chicken, goose,

etc., may often be treated by avoidance of contact. This is not always possible, because of environment, occupation or travel, particularly in the case of individuals sensitive to horse, dog and cat. The technic of treatment for this group is similar to that in pollen sensitization. The results are consistently good and treatment should be carried out when indications and circumstances warrant it.

House Dust.—This can hardly be discussed under the head of specific treatment because of the vagueness of our knowledge concerning its constituents. Our practice is never to use house dust antigen until all other possibilities have been exhausted. The results in those cases treated have been far from satisfactory.

Bacterial.—Wilmer[15] states that after all methods of determining the etiology have failed, then one should classify the case as that of a bacterial type. In such cases, if possible, an autogenous vaccine is made of the polyvalent type, using nasal and sputum cultures. The method of treatment is quite similar to that of most workers, increasing the dose at intervals of five to seven days. Thomas[23] and Touart[24] test the individual with autogenous vaccine prepared from the various bacterial strains isolated, then treat with those to which the individual reacted, frequent doses being given early in the treatment with gradually lengthened interval, followed by prolonged administration at relatively long intervals. Their results have been unusually good. Out of a series of sixty-two cases only eight, or 12.9 per cent, were failures, while approximately 74.2 obtained some measure of relief. Walker,[24] in his early work, advocated the use of autogenous vaccines, using the predominating organism. He claimed most satisfactory results, and in a later work[25] reports 40 per cent of nonsensitive bacterial asthmas relieved and 20 per cent improved. He claims that the permanency of relief is dependent upon the individual resistance to the bacteria in question, therefore the duration of relief varies.

Rackeman[26] states that three theories may account for relief obtained from the use of vaccines: First, that asthma may depend upon low grade infection of the bronchial mucosa. Second, that the patient may be sensitive to bacterial proteins.

Third, that vaccines produce a certain degree of protein shock. In his series of 131 patients treated with autogenous and stock vaccine, all treated on the basis of local reactions, his results were practically alike with both types of vaccines. Our own experience with vaccine therapy has been similar to that of most workers in that our results were equally as good with the use of either stock or autogenous vaccine, and when relief was obtained by their use it was usually rapid and spectacular, but we are very frank to admit that our results with this method of treatment were not as spectacular nor the percentage as great as reported above.

Local and Surgical Treatment.—The treatment of respiratory mucous membrane during acute allergic attacks with ephedrin sulphate, 3 per cent, or cocain HCl with adrenalin gives a great measure of temporary relief, but has no place in attempting to give any permanency of relief.

Operative measures should never be attempted during acute attacks. One had better wait for an interval between attacks during which time whatever surgery may be necessary can be done. We believe that most sinus conditions in the allergic individual are merely coincidental or secondary to the existing allergic condition present, which is in accord with the opinion that, regardless of whether the nasal pathology, such as sinusitis or focal infection in the nose or throat, be secondary to the allergic state, we strongly urge and advise that it be given the proper surgical attention, particularly when drainage and proper ventilation is interfered with. Polypi if present should be removed, not because it is believed that they produce symptoms, for we believe they are the result of the allergic state, but to permit of proper drainage. Broadly speaking, all foci of infection should be eradicated for the general good that the patient will derive from the procedure.

Bronchoscopy in chronic asthmatic bronchitis, bronchospasm or bronchial asthma is of no value; all workers having employed this procedure report no results that warrant such procedure. What relief is obtained from the removal of mucus plugs is very temporary, and what relief is obtained from the instillation of lipiodol can just as well be accomplished by the oral administration of potassium iodide.

The functions of the otorhinolaryngologist in allergic disease may be summed up as follows:

To recognize the characteristic history of allergic disease and to be aware of its frequency.

To recognize the characteristic effect of allergy on the mucous membrane of the respiratory tract and the sequelæ that may arise therefrom.

To realize that the removal of the sequelæ, although frequently necessary, will not affect the course of allergic disease.

To be governed by this knowledge in undertaking any surgical procedure in an allergic individual, particularly those procedures aimed at the removal of possible foci of infection or the clearing up of so-called "trigger areas."

And finally to give symptomatic relief with the full realization that the underlying cause is not thus being influenced.

We do not mean by placing all these restrictions on the otorhinolaryngologist to imply that his place in the care of the allergic individual is a minor one. On the other hand, it is to him that many allergic individuals first come seeking relief. It is to emphasize his responsibility in the proper diagnosis and care of those sufferers that leads us to labor the point as we have done. Surely, at least in the past, our admonitions have been necessary, for in 1925[29] we collected a group of 834 allergic cases and obtained in 413 of them a history of 712 operative procedures on the nose and throat. We felt and still feel that such statistics, in the light of our present knowledge, are unjustifiable, and we are certain that otorhinolaryngologists as a whole will support us in this statement.

## SUMMARY.

1. Allergy, or an abnormal sensitivity to foreign proteins, is a frequent cause of respiratory symptoms.

2. These symptoms may be grouped into syndromes termed hay fever, allergic bronchitis and bronchial asthma of seasonal or perennial type.

3. The symptoms of respiratory allergy are the result of characteristic changes in the respiratory mucous membrane.

4. The diagnosis of respiratory allergy is based on the hereditary history, the characteristic personal history, and physical findings and on protein skin tests.

5. Specific treatment is based on the proper correlation of the findings in all the above procedures.

6. Nonspecific or less specific treatment based on the doubtful theory of the bacterial causation of allergic disease of the respiratory tract is discussed and its marked limitations are stated.

7. Surgical treatment is indicated only to remove the sequelæ of allergic disease or in certain instances to remove foci of infection.

8. Topical treatment for the relief of symptoms is purely a temporary measure.

9. The responsibility of the otorhinolaryngologist in the care and treatment of allergic respiratory disease is emphasized.

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LXI.

# REPORT OF A CASE OF MIXED TUMOR OF THE PAROTID TYPE GROWING FROM THE POSTERIOR ASPECT OF THE THYROID CARTILAGE.

By R. C. Lynch, M. D.,

New Orleans.

I saw Mr. R. first on July 7, 1924. At this time he was suffering from some dyspnea hemoptysis, hoarseness and cough. General history was unimportant except that symptoms came on gradually about six months before and had increased to present state.

X-ray of chest and physical was negative, Wassermann negative and blood study was normal. He had received no injury to account for symptoms.

From examination I noticed a fixation of the right vocal cord, but not complete, with a slight lag in motion of the opposite side. The cords were not red or swollen, and the margins were smooth. The false cords and arytenoids were normal. One could see that below the cords posteriorly there was a rounded smooth mass, as if bulging the posterior wall of the larynx, and the upper two rings of the trachea into the larynx and over the subglottic area posteriorly there were some blood streaks or clots.

With suspension under general anesthesia by pushing the cords aside one saw the same appearance described above except more extensive, and by palpation the area was found to be very firm as if the mass was a chondroma.

A thyrotracheotomy was done to better view the area. The mass was found to be so firm and adherent that dissection removal seemed impossible.

Accordingly a portion was removed from the upper trachea area and ten needles of 5 mg. each were placed in and around the growth. They were left in for six hours.

A tracheotomy tube was placed at the lower end of the wound and a tentative diagnosis was made of chondrosarcoma. The trachea was packed and kept open for observation. After a period of seven days the wound was allowed to heal and breathing was again resumed through the mouth.

Report of the pathologist was mixed tumor of the parotid type. Patient returned home and seemed apparently well, with instructions to return should symptoms return. He had a good voice and functioning cords.

In March, 1927, he returned with the same symptoms, and this time a tracheotomy was done under local anesthesia. Three days following, under general anesthesia and suspension, second regrowth was observed and the thyrotracheotomy wound was reopened.

The tumor extended from behind the thyroid cartilage and apparently densely adherent to the posterior wall of the trachea extending down to the fourth ring. At this time two needles of 12½ mg. each of radium were planted into the growth and 200 mg. of radium were cross fired externally, the latter screened with 1 mm. of brass and a finger cot and placed 3½ inches from the skin. The reaction was moderate and again followed by a retrogression of the tumor mass, but it was necessary to retain the tracheotomy tube for safety.

He was comfortable and talking in good voice and breathing with tracheotomy tube from this time, April, 1927, to February, 1929, when he returned again with the tumor giving about the same symptoms as before. Radium in same quantity, method and time was used.

April 18th, he returned again with protruding mass growing from the margin of the tracheotomy tube, hard, not very vascular, but firmly adherent to its outer wall. The lumen of the trachea was smooth and pink and uninvolved. This mass was removed as completely as possible, which was very incomplete, and two 12½ mg. of radium put into its base for eight hours. The patient returned home.

The specimen was given to the pathologist, who again reported a mixed tumor of the parotid type.

June 6, 1929, he returns again with no evidence of tumor externally. The cords are fixed in the midline, but will vibrate

on talking, which he does very well when the tube is closed. There is more encroachment of a mass on the right beneath the cords and trachea, feels very hard and fixed, for the first time he has a slight dysphagia, and there is still some evidence on the skin surface of the radium application made in April.

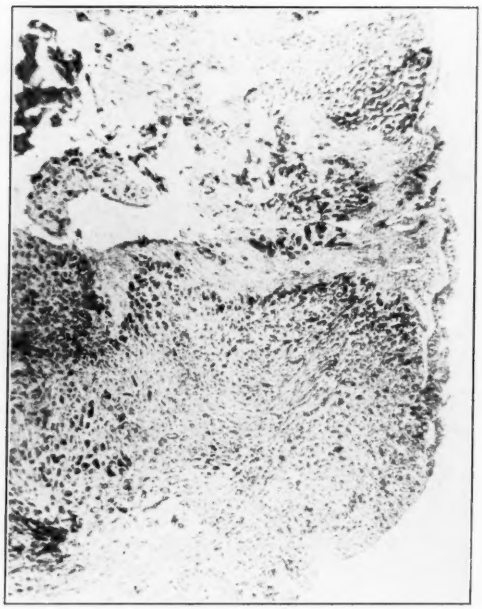
The lantern slides of the tumor and X-ray plates show the pathology and location.

He has willed his trachea to Dr. Shea of Memphis, through whose courtesy this case was referred to me.

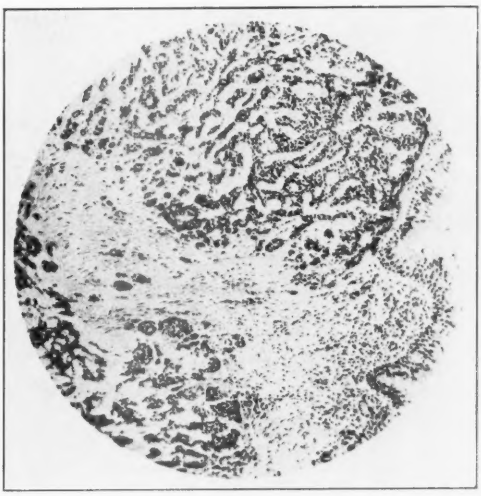
It is unusual to see a tumor of this type growing in this location. The tumor and its pathology differ from an adherent thyroid.

I report it because of its rarity.

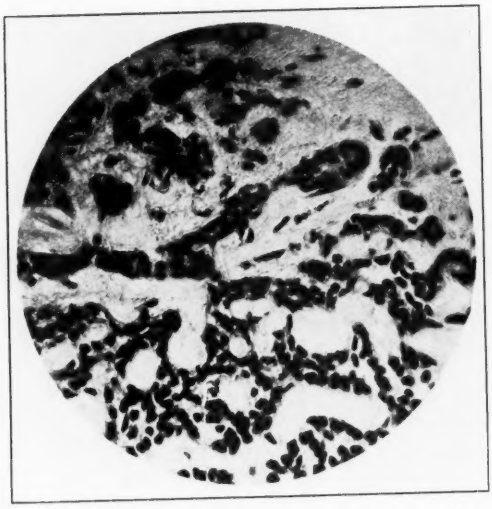
7a. 10x--Pedunculated tumor mass covered with stratified epithelium. Very little stroma noted as white lines.



7b. 30x—Mass of tumor cells showing dark staining nuclei. Scanty stroma. Surface covered with tall columnar epithelium.

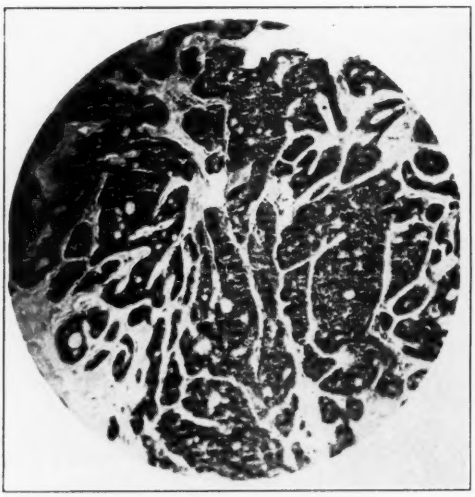


7c. 60x—Cords of epithelial cells surrounded by smooth stroma varying in amount. Mass covered by stratified ciliated epithelium.

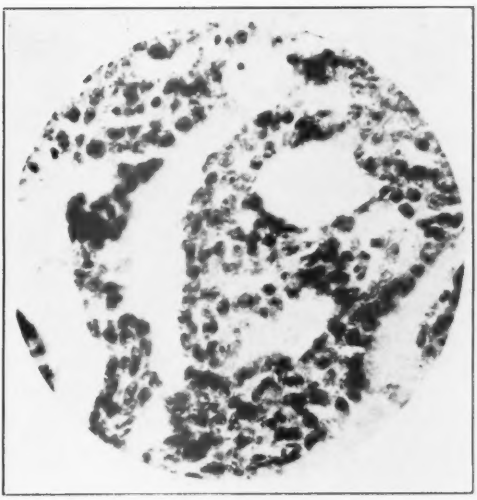


7d. 275x—Irregular groups of epithelial cells attempting to form acini, and surrounded by a myxomatous stroma.

8a. 10x—Section through entire thickness of recurrence. Note layer of skin covering the dense tumor mass which is infiltrating the surrounding tissue.



8b. 60x—Groups of dark staining cells surrounded by a smooth homogeneous stroma containing but few nuclei.



8c. 275x—Actively growing epithelial cells, some in mitasis, surrounded by a smooth myxomatous stroma.

LXII.

# CHRONIC ETHMOIDITIS: ITS CONSERVATIVE SURGICAL TREATMENT.*

By Ross Hall Skillern, M. D.,

Philadelphia.

When one offers a treatise before a gathering of this sort, it is more or less understood and expected that his presentation is rather in the form of a compilation taken from every available authority, and in the end it leaves the listener to draw his own conclusions as to the relative efficacy of the various methods described. Now I propose to deviate from this custom and present the subject entirely from our own point of view; in other words, give it as we see and understand it, with the treatment that we have found most suitable for that particular case or condition.

Chronic infections of the ethmoid, as is now well understood, can broadly be divided into the suppurative and non-suppurative. In the first instance an infection is present characterized by the formation and exudation of a purulent secretion accompanied by the appearance in the ethmoid region of a more or less widespread catarrhal inflammation. The purulent secretion varies from time to time in its consistency, at one time being thin, acrid and almost watery, and again assuming the characteristics of a thick, yellow discharge, the same which the older surgeons were wont to term laudable pus. In the ordinary case, variations between these extremes were the rule rather than the exception, depending upon, as the patient would say, the acquisition of a fresh cold, which usually was not the case, but in reality represented more an acute exacerbation of his chronic condition. This would naturally account for the changes in the consistency of the secretion, which was sometimes thick and profuse, sometimes thin

*Read by invitation before the British Med. Assn., Cardiff, Wales, July, 1928.

and scanty, ofttimes making one erroneously believe that a cure was in progress. As the infection spread, the discharge, particularly that collecting during the night, became more constant, finally resulting in that condition of chronic purulent ethmoiditis which is well known to all of us.

The other form of ethmoiditis, the nonsuppurative type, is a much later development. It was recognized in a way by some of the early pioneers, as, for example, Woakes and Bosworth, but it was not until after Uffenorde published his treatise that it obtained universal acceptance. The type I refer to is that form characterized by polypoid hypertrophies and even true polyp formation unaccompanied by true leucocytic pus. Hitherto, Zuckerkandl's postulate that the polyps were the products of the purulent secretion, had been accepted without question. Even after the publications of the microscopic pathology both in Germany and the United States, much doubt was expressed as to the proper interpretations of these findings, and many months, I might add years, elapsed before this doctrine was accepted at even its face value. I think we all now recognize hyperplastic ethmoiditis as a separate and distinct pathologic entity in contradistinction to the earlier known suppurative infection. The fact that these two can and do occur as separate forms does not preclude the possibility of their being combined and occurring as hyperplastic infection with suppuration. As a matter of fact, the probability of the suppuration is directly as to the extent of the hyperplasia, the greater the polyposis the more likely that the commoner forms of saphrophytic micro-organisms will find a suitable media for their growth with the formation of their suppurative products. When these once gain a foothold I have never seen them disappear.

As the ethmoid labyrinth is not only divided into two portions (the anterior and posterior) by a complete partition, but these also are subdivided into numerous chambers (cells), and as these may be individually affected it would be well before taking up the treatment to make a more comprehensive classification. Following this, the indications for surgery with especial reference to certain methods will be greatly clarified.

Suppurative:
(a) One or more of the anterior cells.
(b) One or more of the posterior cells.
(c) Combined, affecting both anterior and posterior cells.
Hyperplastic:
(a) Localized hyperplasia (small polyps).
(b) Anterior labyrinth.
(c) Posterior labyrinth.
(d) Intra-cellular.
(e) Extra-cellular.
(f) Combined.

The last three, d, e and f, may represent merely an extention of the inflammatory process and can occur equally in any part of the labyrinth.

(g) Suppurative infection.

This represents the final stage when either a new infection occurs or the old one has advanced so far and the tissues have become so infiltrated as to invite leucocytic diapedesis with the formation of a purulent secretion.

Let us now consider individually these different forms.

(a) *Suppuration in One or More of the Anterior Cells* (Fig. 1.)—This intranasal type, so far as I am concerned, as an individual entity with one exception is nonexistent. Of course, when an orbital ethmoid cell situated behind or external to the frontal sinus and outside the nose is present, it may easily become infected and simulate a frontal sinusitis; but as we are concerned only with that portion situated within the nasal cavities this lies beyond the scope of our presentation. The remaining anterior cells (with the exception noted above, which I will come to later), are those known as the infundibular, which includes the frontal bulla, when present, and that one lying within the agger of the nose. I do not recollect ever observing a circumscribed chronic purulent infection of the mucosa of these cells unaccompanied by infection of the frontal sinus. Presumably this is possible, but since it has never come under my own observation nor that of my colleagues, its occurrence is, as far as I am concerned, only of academic interest.

The exception referred to was a purulent infection of the lining membrane of the bulla ethmoidalis. Formerly, an abscessed bulla was considered an event of no uncommon occurrence, but this was probably due to the fact that when it was present it so overshadowed other infection as to cause the latter to be entirely overlooked. I am sure if you review in

your minds the cases occurring in your practices you will bear me out in this statement. However, other things being equal, the evacuation of a badly diseased bulla will bring about a cure, despite the fact that the infection has embraced other cells in the immediate neighborhood.

(b) *Purulent Infection of the Posterior Cells* (Fig. 1).—That these cells are more frequently the seat of chronic disease is due to a number of conditions. In the first place, they are larger and more numerous. Secondly, they are so situated as to be less amenable to aeration due to the presence of the middle turbinate. Their drainage can easily be interfered with by slight swellings of the nasal mucosa, and lastly, their anatomic situation prevents easy access to exact diagnostic instrumentation, even with the aid of the nasopharyngoscope. Being but slightly disturbed by the inspiratory air currents they offer a fine media for microorganismal growth once infection has taken place. This extension may be so gradual and insidious that the patient is unaware of the seriousness of the condition until a well marked infection has occurred; he having, it is true, noted the unwonted postnasal discharge and stuffiness in the nose, particularly on arising, but as the inconvenience was not great, putting the whole matter down as a particularly tenacious cold. This, however, is noted more in the hyperplastic variety than in the suppurative.

(c) The combined form affecting both the anterior and the posterior cells is always more frank in its course, as it usually represents (in contradistinction to the other types) the direct sequelæ of an acute infection or perhaps a series of acute inflammatory disturbances. In the event of such a widespread infection, the symptoms are so apparent as to obviate further elaboration.

*The Hyperplastic Types.*—This, of course, is a pathologic condition. A portion of the ethmoidal mucosa undergoes hyperplastic changes, which range from slight degeneration in a small portion of the lower border of the middle turbinate to the formation of the actual sessile polyps the size of a small oyster. While the lower edge of the middle turbinate appears to be most frequently affected, nevertheless the actual polyps,

when present, appear to take their origin higher, having their bases in the body of the ethmoid or along the uncinate process. I do not recalled ever having observed large single polyps originating along the lower edge of the middle turbinate. The anterior labyrinth is usually the seat of the large extracellular hypertrophies which lie more or less loosely in the nasal cavities, while in the posterior labyrinth the intracellular variety hidden from view is the rule. This is probably due to the fact that there is more room for growth anteriorly beneath the turbinate than posteriorly between the body of the ethmoid and the nasal septum. The large so-called choanal polyps do not, as a rule, originate in the posterior cells, but if traced to their source will be found to spring from the mucosa of the maxillary sinus, forcing their way out through the *pars membranacea* and extending into the nasopharynx through the medium of a long pedicle. Of course, in either labyrinth the primary affection, whether it be intra- or extra-cellular, may become combined, which represents in either case only an extension of the pathologic process from within outward or without inward, as the case may be. This is well illustrated in those old chronic cases where the entire ethmoid, from the uncinate process to the anterior sphenoidal wall, is one mass of polypoid degeneration. All of us have seen such cases which continued for years accompanied by a profuse watery secretion which would dry on the handkerchief, causing a stiffness, but not staining the linen. When infection by the *staphylococcus aureus* occurs the secretion becomes thick and yellow, with the characteristics of true pus, but this seems to appear in cycles, particularly on the acquisition of every fresh cold. The true combined form becomes permanent only when the microorganisms find lodgment deep in the mucosa. Even then the suppuration appears to be different from that caused primarily by the influenza bacillus; for in the first instance the secretion seems to be more profuse and is easily discharged, the nasal passages are comparatively clear, while in the latter the pus is thicker, more tenacious and exhibits a great tendency to form into crusts.

Permit me now to digress for a moment to give you the various forms of treatment used by us and later to apply them to their appropriate pathologic condition.

TREATMENT.

The successful treatment of all sinus infection, particularly where the ethmoid is concerned, has for its basic principles aeration and drainage with emphasis upon the former. We have long since felt that the old dictum of removing all infected ethmoid tissue, irrespective of the kind and extent of the inflammatory process, was as unnecessary as it was radical. With proper assistance, Nature will do much to bring about a resolution of the mucosa in diseased cells after proper aeration has been established. This tendency of Nature is of no little import when we call to mind some of our earlier ethmoid cases which were in the beginning perhaps mild, but, after our well meaning surgical intervention, were transformed into a disorganized suppurating mass with landmarks obliterated and with, instead of relief, augmented subjective discomforts. Observing these results, not only in my own practice but in that of my colleagues, I was prompted to seek the solution and found that over-enthusiasm was the cause of my undoing. Instead of sending a boy to do a man's work, I went to the other extreme and sent a man to do a boy's. In other words, I was prone to overdo the removal of ethmoid tissue rather than conserve it, thinking that to leave even the slightest amount of diseased mucosa behind would result only in a nidus from which the infection would gradually spread until the condition became as bad or even worse than before the operation.

In those days drainage was our deity, the *sine qua non* of a successful treatment. Ventilation, it is true, was looked upon as a desirable but not necessarily an indispensable adjunct. A suppurating cell should not only be opened, but completely eradicated, encroaching upon healthy neighboring tissue if necessary in order to bring about our purpose. Now, thanks to our experiences with the sphenoid in which we saw hyperplastic tissue almost filling the cavity shrink down in a few days under our very eyes until in a short time it was again normal, all this has changed. We know and confidently expect that Nature, with a little assistance, will do a far better and cleaner job and procure a physiologically functioning area of tissue in the ethmoid region, while we with our wide-

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spread and ofttimes haphazard procedures only leave an area of débris which soon becomes reinfected and degenerates into a disorganized fibrous suppurating mass.

It is to anticipate this very eventuality that I propose to lay before you for your consideration the various forms of treatment practiced by us at the present time. It is not in the form of a recommendation, but rather as a communication that these are presented, not to be taken as dogmatic in any sense, but rather at their face value for what they may be worth.

Let us begin with the mildest of the suppurative types and ascend until we have the severest complicated form to deal with, applying their appropriate treatment as each is met.

*Simple Catarrhal Infection.*—This is characterized by mild headaches, stuffy nose and a fairly constant thin mucoid discharge, which becomes thick and yellow at times, the patient being apparently very susceptible to attacks of acute coryza. Rhinoscopic examination often shows but little besides a hyperemia and congestion of the mucosa when compared with the opposite side. Pus is conspicuous by its absence, at least as far as the examiner is concerned. However, the patient often states that considerable discharge, particularly postnasal, is in evidence during the morning hours. This form yields slowly to the old shrinking and cleansing treatment, but shows a great tendency to recurrence. To obviate these, I have found the removal of the swollen and often infected middle turbinate almost a *sine qua non*. The purpose of this is first of all to permit thorough aeration and ventilation to the diseased cells, and secondly to furnish sufficient space to make possible the application of cotton poultices which are saturated with certain medicaments. In given cases where the nasal passages are congenitally narrow and the mucosa of the septum is greatly thickened, I have found it necessary to resect that part of the septum in its entirety, making a huge perforation so that these membranes can no longer swell and compress the membrane of the ethmoid on its respective side.

After the middle turbinate is removed and the roughened edges are smoothed nothing further is done, even though the temptation be severe, and the parts are allowed to heal. After five days have elapsed treatment by means of tampons is instituted. This consists of the application of a cotton tampon

(Fig. 2) saturated with a 10 to 20 per cent solution in half water, half glycerin, of a nonirritating silver preparation which covers the ethmoid from the anterior sphenoidal wall to the uncinate process. This should be allowed to remain *in situ* at least five hours. I use a preparation called silvol, but there are others which to all intents and purposes are identical. These applications deplete the tissues of their inflammatory content by the hygroscopic action of the glycerin, thereby permitting the silver partially to penetrate the cellular interstices and bring into action its germicidal and astringent effects. In order to obtain the maximum effect three things are essential: (1) The solution should be freshly made; (2) the solution should be warm; (3) the tampon should remain in place five or more hours. These treatments are instituted at first every other day, gradually tapering off directly as to the improvement noted. This usually requires but a few weeks, as the affection readily yields to this form of treatment.

*Purulent Infection Confined to the Anterior Cells.*—As a disease *per se*, this is one of the rarest we are called upon to treat. It is true that occasionally one sees an infected and suppurating bulla ethmoiditis and even an orbital ethmoid cell which simulates a frontal infection, but by and large when the anterior cells are suppurating we have an infected frontal sinus which is also secreting pus. Under these circumstances a cure of the underlying ethmoid depends upon first ridding the frontal of its infection. Sometimes after this is accomplished the bulla will continue to secrete owing to the ostium's becoming partially occluded from the inflamed and swollen mucosa, thereby seriously interfering with the normal drainage and practically eliminating aeration. When this occurs it will be necessary to open the floor of the bulla (Fig. 3) and reinstall both drainage and aeration. If this is the sole remaining focus of infection, a cure speedily follows.

*Suppuration in One or More of the Posterior Cells.*—This condition, although not uncommon, is one of the most difficult and one of the most rarely early diagnosed of the sinus infections. The reason for this is apparent when we consider the mild and ofttimes vague symptom complex together with the almost inaccessible location, even to the nasopharyngoscope, of these posterior cavities. We can study with a fair

degree of exactitude the cells emptying into the superior nasal passage, but those which lie to the outside of these and those lying in the depth of the sphenoethmoid fissure are beyond our inspection. Indeed, many of these cases, with a low grade infection in one or more of the cells, have escaped detection for years, having been considered merely a chronic catarrhal condition, until the sudden appearance of a severe ocular disturbance invited immediate attention, which then resulted in the discovery of a latent sinusitis. One clue is always at hand if we are sufficiently astute to take advantage of it, and that is the appearance of the middle turbinate. The body of this structure appears slightly hypertrophied and ofttimes has a mottled appearance. While to the casual observer this may escape notice, yet if one carefully compares the turbinate with its fellow on the opposite side, the difference will at once be apparent.

The logical treatment is, of course, to secure better aeration and drainage, which can be accomplished only by the total removal of the middle turbinate. This resection not only aids materially in contributing to these therapeutic advantages, but incidentally brings under our view those cells which subsequently may require opening. Unless the suppurating areas have accurately been disclosed or unless symptoms are pressing, it is well to wait for a few days in order definitely to determine the exact seat of the infection before attacking these cells. The silver poultices will be of great advantage in this determination, for I have often seen an apparent general suppuration under this treatment localize itself into one or two defined regions which lent themselves admirably to instrumentation. Formerly it was our practice completely to excavate all these posterior cells when infection was discovered in their midst, but now we open with the hook, and with the Grunwald forceps remove only the diseased portion until healthy tissue is reached. In this way we have not only procured quite as good end results, as far as a cure was concerned, but conserved tissue, which certainly possesses a normal functioning activity. After these cells have been opened and exposed to thorough aeration, Nature speedily effects a cure which is in direct relation to the thoroughness of the operation.

*Combined Suppuration Affecting the Entire Labyrinth.*—This condition is usually an old one, where the middle turbinate has long since been sacrificed, but from one cause or another the infection has gradually spread until both the anterior and posterior cells are more or less involved. Nothing but a so-called complete exenteration will be of the slightest permanent avail in these cases. In our experience the crux of this situation lies in the preliminary removal of the uncinate process (Fig. 4), for by this step we gain from ⅛ to 3/16 inch room, which may prove of inestimable value in reaching the agger and infundibular cells, to say nothing of the depths laterally to the sphenoethmoid recess. This can readily be accomplished with a flat chisel without particular danger in careful hands.

After the uncinate has been removed, the posterior lying cells may be methodically attacked and resected, keeping close to the orbital plate until the anterior sphenoid wall is reached. It is well to leave a certain amount of the inner wall of the ethmoid *in situ*, particularly that portion which houses the terminal filaments of the olfactory nerves (Fig. 5). While it has been shown that infection may travel through the cut sheaths of these nerves and enter the meninges, nevertheless I do not fear this so much as I desire the preservation of the sense of smell in this locality. Regarding the occurrence of meningitis following the severance of these nerves, it must be a very infrequent occurrence, otherwise all of us would at one time or another have met with this tragic misfortune. Even though I have had fatalities, I do not believe any of them has been due to this cause. As a matter of fact, I have on several occasions removed diseased bone which exposed the pulsating dura in the immediate vicinity of the olfactory fissure, which in one or two cases was followed by the escape of a quantity of cerebrospinal fluid. Most of these, however, despite my anxiety, recovered without untoward symptoms. Notwithstanding these fortunate outcomes, it is nevertheless better to be safe and shun this dangerous region by conserving the olfactory area with its mucosa, as ventilation and drainage should obtain quite as well as though it had also been removed. Following this procedure I know of no condition

that reacts more favorably to the silver tampons as an after-treatment than this one.

*Hyperplastic Ethmoiditis, Localized Hyperplasia (Polyps).*—The surgery of polyposis and polypoid condition affecting the ethmoid is of such universal knowledge that I do not propose to abuse your patience by detailing these procedures. I shall, however, touch upon some of the more prominent facts concerned in the permanent ablation of these growths. The localized hyperplasias or separate polyps springing from the nasofrontal region and beneath the anterior end of the middle turbinate are usually removed with the snare. Unless, however, the bony attachment is also resected, they are almost certain to return; therefore, emphasis should be placed on this phase of the operation in order to insure a permanent disappearance of the growths. The preliminary resection of a portion of the middle turbinate depends upon the accessibility of these bases to instrumentation. I have often noted in the presence of these moderately large sessile polyps that the hyperplasia is usually confined to the mucosa outside the cells, it being rare to find coexisting intracellular polyps. The one exception to this is the old chronic cases where the entire labyrinth has long since been diseased. While polyposis of the anterior cells is nearly always extracellular, with the posterior cells the opposite obtains, as one rarely encounters large polyps hanging down into the choana which take their origin from this region. (The large solitary choanal polyp in nearly every instance comes from the maxillary sinus, a fact which I have been able to demonstrate time and time again.) This does not mean that the external mucosa is not also affected, for it, too, shows marked change, but it is more in the nature of a polypoid degeneration than of large pedunculated polyps.

When the anterior and posterior labyrinths are coaffected we, therefore, have both an intracellular and extracellular condition to deal with. As a rule, these combined cases have progressed insidiously over a number of years until the entire ethmoid capsule has become a mass of polypoid degenerating mucosa. This is the type of case which is often associated with asthma and hay fever, or at least its symptom complex may be attributed to the presence of the polypoid tissue. We have found that the only rational treatment from a curative

point of view is the complete removal, as far as is humanly possible, of the polypoid degenerating tissue.

Several methods lie at our disposal, and we always try to select that one which seems to be particularly suited to the individual case. In this way exigencies, such as abnormal anatomic configurations, are met with and successfully overcome. Ignoring, then, the ordinary procedures adapted to the simpler conditions, we shall consider those commonly used in our country when complete exenteration of the ethmoid is under consideration. These are the Grunwald, the Hajek, the Mosher, the Sluder and the Ballenger in their various modifications. Most of these methods, in their original concept, have long since become obsolete, so I shall only briefly picture the various steps as now practiced, at least in our clinic.

## CONCLUSIONS.

A brief retrospect over the intranasal methods of operating upon the ethmoid shows us first of all that to accomplish successfully any operation that is worthy of the name an intimate knowledge of the anatomy, both relational and surgical, is essential. This knowledge, like a two-edged sword, cuts both ways, for it not only gives the operator confidence fearlessly to remove tissue, but also the wisdom to exercise caution when approaching a dangerous area. All of us who have concerned ourselves to any considerable degree with intranasal ethmoid surgery must have been convinced that it is impossible in every case completely to expose the mucosa of each and every ethmoid cell. At least, this holds true in my own experience. There exists, unfortunately, no anatomic configuration of these cells that one can use as a normal model, so that in operating (despite all the stereoscopic skiagraphs we may have had made) it is the idealistically normal that we must keep in mind, and if in the midst of the exenteration we encounter great variations, they must at once be considered anomalous and we must proceed with the operation accordingly. Small wonder is it, then, that certain deep-lying cells escape our attention, this being the reason why in these cases our ultimate results are not 100 per cent good. Indeed, it is now my custom to try to impart this knowledge to those patients of better understanding before attempting any operation

and state that the improvement should be measured by a certain percentage, according to the severity and condition of the case, say from 60 to 95, 100 per cent representing, of course, the perfect result. While all of my cases have by no means been restored to their original normal functioning activities, nevertheless I have long since become convinced that, properly executed, the intranasal operation on the ethmoid offers the least dangerous, if not most facile, opportunity to the patient for relieving him of his distressing and ofttimes dangerous infection.

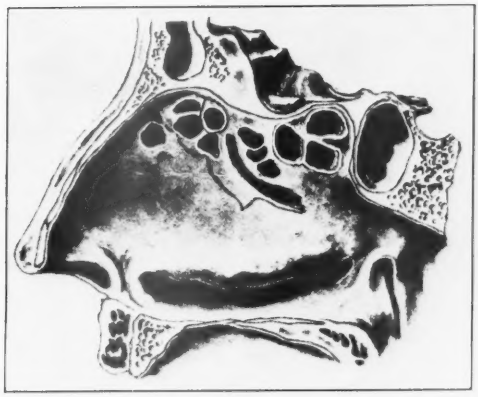

Fig. 1. Anterior and posterior ethmoid cells opened—Hiatus semilunaris divides.

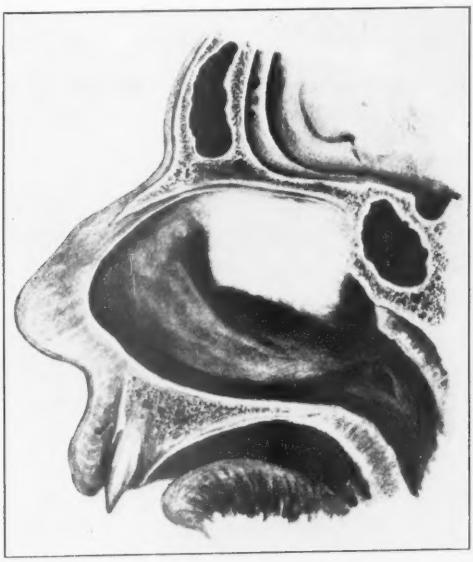

Fig. 2. Cotton tampon applied to ethmoid after removal of middle turbinate.

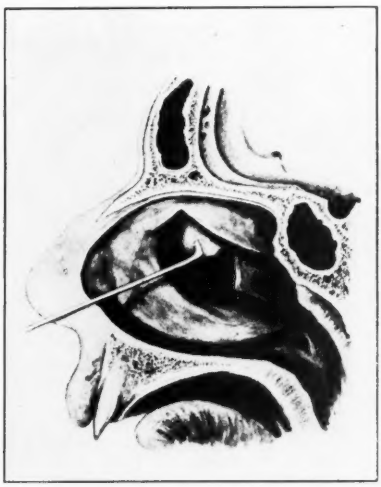

Fig. 3. Opening ethmoid bulla after removal of middle turbinate.

Fig. 4. Removal of uncinate process with chisel.

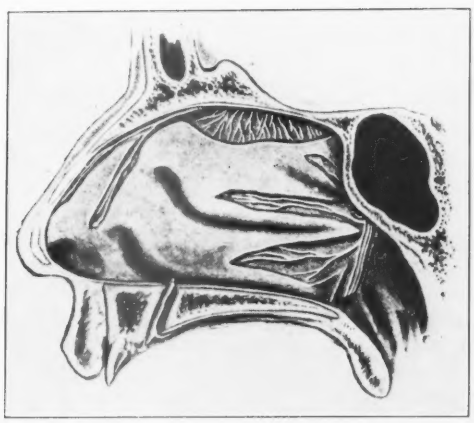

Fig. 5. Nerve supply of lateral nasal wall.

LXIII.

# HISTOPATHOLOGY OF SINUSITIS AND MASTOIDITIS.*

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The gross pathology of sinusitis and mastoiditis is too well known to necessitate repetition in a treatise of this character. But in order to appreciate the significance of these more apparent changes a thorough understanding of the underlying histopathology is essential.

It is my purpose, therefore, to discuss here those pathologic changes which occur in the individual tissues of the nose, the paranasal sinuses and the mastoid as a result of infection or irritation, and not to include tuberculosis or malignancy. These latter conditions are not locally characteristic, but assume the microscopic appearance of similar processes elsewhere in the body. These descriptions represent the careful microscopic examinations of more than 300 specimens removed at operation, for definite clinical reasons, from a large variety of cases at Barnes and St. Louis Children's hospitals.

I hope to convey a clearer understanding of this subject by presenting, first, the changes which occur in the nasal cavity, and then the actual changes which occur in the respective sinuses themselves, the nature of the pathology in the tissues of the nose being so often pathognomonic of that in the sinuses.

## ENLARGEMENTS OF TURBINATES, INFERIOR.

*Hypertrophy*, as seen in the clinical condition known as hypertrophic rhinitis, or intumescent rhinitis, giving rise to bilateral, unilateral or alternating stenosis of the nasal pass-

*Candidate's thesis for the American Laryngological, Rhinological and Otological Society, July 4th, 1929.

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ages, was found on microscopic examination to be an engorgement of the blood spaces accompanied by an enlargement of the already existing connective tissue cells of the tunica propria, without an increase in their numbers. There was not usually an extravasation of white or red blood cells. There were no bone changes. This type of turbinate retains its elasticity and shrinks down under the usual astringents. It is the result of a physiologic rather than a pathologic process. The glands are usually dilated and hyperactive, though not increased in number.

Hyperplasia.—This is a later stage of an hypertrophy. The epithelium was thickened, the tunica propria more or less densely infiltrated with connective tissue cells of all ages, and with lymphocytes, the walls of the blood spaces thickened, the bone undergoing degenerative and regenerative changes and fibrosis. The glands were mostly compressed, others either dilated or confluent. Such a turbinate has lost its elasticity and contractile power and does not shrink down under the action of the usual astringents. Hypertrophy and hyperplasia were found coexistent in the same turbinate.

*Polypoid Degeneration.*—The type that is seen at the posterior extremities of the turbinates and designated as mulberry hyperplasia presents the following microscopic picture: Reticular tissue with marked young connective tissue cell infiltration and beginning fibrosis in central portion; large blood-filled spaces present. This was completely enveloped, except on one surface at line of severance, with a stratified squamous epithelium which was moderately thickened. The superficial polypoid degeneration recognizable macroscopically along the inferior border of the turbinate was similar in appearance to the above, except that the large blood spaces were not present.

MIDDLE TURBINATE.

*Epithelium.*—The epithelium of this turbinate, which is normally stratified and columnar in type, was found to have undergone thickening by one or all of three ways, namely, the multiplication of the layers of columnar cells, the distension and prolongation of goblet cells or the metaplasia of the columnar into stratified squamous epithelium. This flattening of the cells was limited either to the two or three more

superficial layers, or it involved all of the layers down to the basement membrane (Fig. 1). Metaplasia seemed to occur more often at the anterior end and at points of convexity of the turbinate on the medial aspect, leading us to believe that it was in response to direct irritation caused by dust particles, smoke and gases carried by the inspired air, rather than to any circulatory or chemical phenomena within the underlying tissue. It was always found at points at which the epithelium would be most susceptible to inhaled irritants, and did not occur over a large expanse of turbinate generally.

Epithelium which was undergoing disintegration was characterized by a ragged appearing line of cells or a single row of nuclei on a thickened basement membrane. This process of disintegration was frequently accompanied by an infiltration of lymphocytes and microorganisms, but more often it appeared to be the result of friction caused by the contact of the turbinate with the septum, or with the lateral wall of the nose. There was also present a nonstaining layer of mucus on the cells, occurring more frequently along the posterior half and the medial aspect of the turbinate in regions which had been subjected to the constant moisture of irritating secretions flowing over them from the upper sinuses. While it is difficult to associate any of these changes with a specific clinical condition, as they may, and usually do, coexist in any long standing intranasal infection, the mucoid changes seemed to be most apparent in the patients with asthmatic symptoms or in those prone to frequent acute exacerbations of a low-grade chronic infectious process.

*Basement Membrane.*—This structure, called by some the membrana propria, could invariably be differentiated from the tunica propria in the turbinates, being found extremely thickened in some specimens and moderately so in most of them. It usually retained its homogeneous pale pink appearance and was frequently infiltrated by bacteria or lymphocytes. The thickening was most marked when the epithelium was sloughed, the membrane assuming a fibrous-like aspect. The thickening of this subepithelial layer in so many specimens, especially when the epithelium was sloughed, lead us to ascribe to it a secondary protective function for the more deli-

cate structure of the tunica propria against both mechanical irritation and invasion of bacteria.

*Tunica Propria.*—The three outstanding features of this stratum of the mucosa were the edema, the fibrosis and the round cell infiltration. The edema was more noticeable in the superficial portions in both the turbinates and the cell walls, and usually most pronounced at the anterior extremity of the former, though it frequently involved the entire tunica down to the periosteum, being invaded by strands of connective tissue in the deeper portions. In a great many specimens the connective tissue was more pronounced in the areas adjacent to the bone, extending outward until the tunica consisted almost entirely of dense fibrous tissue. (Fig. 2.) The cells, including lymphocytes, polymorphonuclear leucocytes, young connective tissue cells, and at times large numbers of eosinophiles, while invading the entire tunica, more often concentrated around the glands. This infiltration varied in intensity. The picture of lymphocytic reaction around the mucous glands led to the belief that these were the sites of the greatest chronic inflammation and the greatest activity in combating infection. This fact was borne out by the increase in secretion of groups of glands when other groups had undergone pressure atrophy. The mucus glands were either dilated, this dilatation at times being sufficiently extreme to cause a rupture of the walls and a confluence of the glands, or the glands were compressed by the edema or the fibrosis. The extreme dilatation of some glands could also be ascribed to a backing up of secretion, due to their inability to drain sufficiently rapidly as a result of compression of their ducts, causing a transudation of mucus into the tunica propria, thus establishing a condition of extensive polypoid change. The individual gland cells of the acini and tubules likewise showed certain changes, being compressed by the intraacinar or the intratubular pressure, respectively, or dilated by intracellular pressure, thus obliterating the lumen entirely. All of these processes resulted in a disintegration of the gland and tubule. To compensate for this, numerous small new glands seemed to be developing in other areas and appeared to be surviving or perishing, according to the amount of pressure which they were able to withstand.

*Thickening of the Walls of the Blood Vessels.*—The blood vessel walls were thickened, some very little, others a great deal, the thickening in some cases being so extreme that the lumen was almost entirely obliterated (Fig. 3). In some instances the vessel wall was apparently undergoing a vacuolar degeneration. This thickening was one of the most constant pathologic findings, and for that reason it was considered one of the most significant. According to Rohde,[1] by diminishing the blood supply to a part, connective tissue formation is stimulated, especially in bone undergoing repair; consequently, reasoning "a posteriori," since connective tissue cells of all ages were so consistently found, they must have resulted to a great extent from a diminished blood supply, and the interference with the blood supply must have occurred primarily at a point at which the vessels converged, or in the sphenoethmoid region. Extensive true hyperplasia of a turbinate may be, therefore, indicative of hyperplasia, or of even more extensive pathologic changes in the ethmoid capsule and probably in the sphenoid as well, though not necessarily. From the foregoing pathologic findings, it is seen that the changes in the tunica propria of the turbinate and ethmoid usually always go hand in hand.

*Periosteum.*—This structure varied from the normal to a very extreme thickness, and usually in direct ratio to the degree of adjacent fibrosis of the tunica (Fig. 2). At times it appeared actively inflamed and infiltrated with red blood cells, again it was invaded by lymphocytes or young connective tissue cells, migrating from the tunica to the bone. In a certain number of instances the periosteum was scarcely demonstrable.

*Bone.*—Five marked changes were found in the bone: Rarefaction, necrosis, osteomalacia, fibrosis and hyperostosis.

Rarefaction was designated as such only when osteoclasts were found to be actively destroying bone. Osteoclasts occurred either singly, causing isolated erosions along the edge of the bone, or in large numbers, causing a massive destruction from all sides. The potential osteoclast was scarcely ever characteristic — i. e., distinguishable morphologically from the young connective tissue cell or the osteoblast (Fig.

4). Occasionally, it appeared as a large stellate, multinucleate cell, but this was the exception rather than the rule. At times it required the most careful study to determine whether a cell was destroying bone or building it up, and in many cases we were unable to reach a final decision. Just as the action of the osteoclast was the determining factor in rarefaction, the action of the osteoblast was the criterion of hyperostosis. The osteoblasts, when characteristic, appeared as a line of dark staining rod, or short spindle shaped cells lying end to end, parallel to the surface of the bone underneath the periosteum, or to the curve of the lacunæ. The bone surface was smooth and regular and showed an excess deposit of calcium in various stages of enveloping of the osteoblasts, these being enveloped in much closer approximation than occurs in normal bone building (Figs. 2 and 4). This we termed pathologic hyperostosis. The regular arrangement of the osteoblasts contrasted with the ungoverned, irregular invasion of the osteoclasts and was another means of differentiation.

The osteoblasts appeared to originate from two sources, the periosteum and the nonspecific connective tissue of the tunica propria. The osteoclasts originated from the mass of young connective tissue cells, being transformed as they came into contact with the bone. This metamorphosis was only functional, and not physical. According to Von Gaza,[2] paraplastic substances which are released by the dying or dead bone cause this transformation, since necrotic and formative bone stand in such close physical and chemical relationship. However, the latter statement was not always found to be true, since bone necrosis was present in many of the specimens without any attempt at repair by the formation of new bone or connective tissue in the immediate vicinity. The osteoblasts likewise assumed their specific function in certain cases from the young connective tissue cells of the tunica and only became differentiated as they were in the act of being enveloped by calcium.[3] The true osteoblast in most cases came from the connective tissue of the periosteum. Bancroft[4] also says that the periosteum forms an adjunct, but that it is not the only form of connective tissue capable of bone repair. It is difficult to agree with Rohde in his denial

of the metaplasia of the connective tissue, either of the periosteum of the endosteum or of nonspecific connective tissue cells of the neighborhood into osteoblasts, inasmuch as I found this condition in many specimens. In the marrow spaces young connective tissue cells were seen to be definitely assuming the function of osteoblasts and osteoclasts. (Fig. 2.) I am inclined to agree with Louis Christophe,[5] that the chemical process in the blood serum is mainly the basis of new bone formation. This is in accord with the hypothesis that the depression of the blood supply, following thickening of the vessel walls, lead to rarefaction and bone absorption and ultimately to fibrosis.

Necrosis was considered such when the bone took an irregular stain, having a rough, atypical appearance, the nuclei being absent. It is distinguishable from rarefaction in that it is usually an acellular mass action occurring centrally, while rarefaction occurs peripherally and is cellular. Osteomalacia or young connective tissue cell invasion of spontaneously decalcifying bone is a process separate and distinct from either rarefaction or necrosis, being more akin to fibrosis in its earliest stages. It presented a picture of large masses of young connective tissue cells invading poorly staining or nonstaining bone, in which the ghosts of the bone cells could still be seen. (Fig. 5.) The connective tissue cells had apparently swarmed through the periosteum and into the decalcifying bone and completed the resorption of the calcium. Osteomalacia showed the calcium disappearing first and bone cells last, while in necrosis the cells disappeared first and the calcium last. In rarefaction the process was much slower, the bone remaining normal until the osteoclast destroyed it.

Fibrosis always closely followed rarefaction, so that the erosions were quickly filled with connective tissue, which extended in from the periosteum or from the connective tissue of the tunica. (Fig. 2.) In many specimens bridges of connective tissue completely separated particles of bone or islands of bone were found completely surrounded by connective tissue. These may have been instances of heteroplastic bone formation—that is, the formation of new bone separate from the old bone and periosteum, though in the same structure.[6] (Figs. 3 and 6.)

These bone changes have a direct bearing on the aforementioned vascular changes, so far as the extensive rarefaction and fibrosis are the results of diminished blood supply. As Carl Rohde says, after his exhaustive investigations on bone regeneration:[1] "It is thus seen that through the entire question of bone regeneration the important and determining rôle of the vascular system runs like a thread of red. With its preservation and capacity for regeneration, the whole problem of regeneration stands and falls." The vascular changes are widespread and profound, and furthermore, are permanent.

*Atrophy of Turbinate.*—Microscopically this condition was but an advanced stage of hyperplasia resulting in an almost completely fibrosed bone surrounded by dense fibrous tissue, in which were seen larger blood vessels with greatly thickened walls, a few small compressed blood capillaries, many compressed practically obliterated glands, an occasional slightly dilated gland and a few nerve trunks. It is essentially a functionless structure. (Fig. 3.)

SUPERIOR TURBINATE.

Practically the same type of histopathology is apparent here as in the middle turbinate.

EXTRUSIONS OF MUCOSA.

The most commonly met with pathologic entity of this nature is the polyp, the mucus and the fibrous types.

The "mucus type" was composed of reticular tissue, the interspaces of which were filled with homogeneous pale staining myxomatous material. There was a variable amount of round cell infiltration and usually many eosinophiles. (Fig. 7.) Many small, thin walled blood vessels were seen near the surface, which was covered with a stratified columnar epithelium that had undergone metaplasia into squamous epithelium in places. A mucus polyp which had been exposed to radium and removed after about three weeks, showed a marked invasion of the myxomatous zone with young connective tissue cells and fibrous tissue. The epithelium had undergone metaplasia into a stratified squamous type. It was

partially sloughed in places. The basement membrane was not recognizable. (Fig. 8.)

The fibrous polypi, fibromata, were of a different nature, being composed of large numbers of connective tissue cells of all ages, many lymphocytes and many small blood vessels. They are usually attached to the periosteum and appear in the posterior nares of the nasopharynx.

The thickenings of the mucosa of the septum, the lateral walls and the floor of the nasal cavities are microscopically true hyperplasias, as described under turbinates.

### SINUSES.

The gross pathologic changes seen in the sinuses are essentially the same as those seen in the nose, from the simple vascular engorgement to the advanced polypoid degeneration, with the aggravating factor of retained secretions, according to the type of infection, either within the tissues or in the sinus cavities proper. Though there is a great deal in common between the microscopic changes in the mucosa of the respective sinuses, for the sake of clarity I shall describe the histopathology of a composite picture of the various degrees of inflammation found in the individual sinuses.

### MAXILLARY.

The epithelium, which was stratified columnar in type, was thickened by an increase in the number of deeper cells or by an hydropsical process in the cells themselves. There were no true goblet cells seen. It was infiltrated by polymorphonuclear leucocytes or eosinophiles. Again it was seen in various stages of disintegration or entirely sloughed. Metaplasia was not seen. The basement membrane was in some cases not distinguishable, in others much thickened and infiltrated with polymorphonuclear leucocytes. The tunica propria was slightly edematous or had undergone marked polypoid degeneration. The acuity of the process was made apparent by the large numbers of free red blood cells, polymorphonuclear leucocytes and young connective tissue cells, and by the engorgement of the blood capillaries. The more chronic processes were apparent by less engorged blood vessels, thicker vessel walls, fewer red blood cells, older connective tissue cells,

fewer polymorphonuclear leucocytes, more numerous lymphocytes and less marked edema. The glands in the tunica propria were hyperactive and dilated or confluent in the acute and subacute, or later compressed in the chronic fibrosed tissues. This proceeded in some instances to a complete fibrosis of the tunica. The periosteum was not thickened. The bone changes were not so marked, even in the long standing cases. There were only occasional small areas of rarefaction or slight tendency to hyperostosis and fibrosis.

*Frontal Sinus.*—The typical epithelium was composed of a basal layer of round or slightly oval cells and a second zone of round, oval or irregular shaped cells of about the same size. Between these latter occurred rod or pillar shaped cells of about twice the length of the basal cells, singly or in groups of two or three parallel to each other. In certain areas in which the epithelium was thickened, these pillar cells occurred in two to six layers irregularly arranged. The goblet cells were prominent. The surface was covered with a deeply staining pink material, which was apparently the prolonged protoplasmic elements of the individual cells. On the surface of this substance there was a ragged appearing irregular staining border varying in thickness, the ghosts of the cilia. (Fig. 9.) This general picture was deviated from in many specimens by a complete absence of epithelium or an absence of all but the basal cells. In others in which the cilia were more distinct there was a straight, clear cut, dark staining line to which they were attached at their bases. The basement membrane was not, as a rule, distinct from the submucosa, though at times it was much thickened. The tunica propria was extremely edematous and noticeably infiltrated with young connective tissue cells and polymorphonuclear leucocytes, especially around the glands. The glands themselves were dilated and confluent or were partially disintegrated, the basal nuclei remaining. There was moderate connective tissue increase, a much thickened periosteum and moderately thickened vessel walls. There were also a few thin walled engorged vessels. The bone showed generalized calcium absorption only.

*Sphenoid and Ethmoid Sinuses.*—I have been unable to distinguish any difference at all between the sphenoid and

the ethmoid mucosas except that the former may be slightly thicker. I shall, therefore, describe them as one.

*Epithelium.*—Stratified columnar, composed normally of but one to three layers, besides the basal layer, which resembled that found in the epithelium of the frontal sinus. While the epithelium was moderately thickened in most cases, this was due in the majority to the superficial polypoid changes rather than to a direct multiplication of cell layers. In very few cases were the changes in this epithelium as profound as in that of the turbinates. Metaplasia was not observed. The cilia were either barely discernible or entirely absent.

*Basement Membrane.*—This was not always demonstrable, though in a few cases it was definitely thickened. It was in some specimens infiltrated with polymorphonuclear leucocytes.

*Tunica Propria, Periosteum, Bone.*—The changes in these strata followed the general trend of the changes found in the middle turbinates as described above, though to a lesser degree, as a rule.

MASTOID.

*Adult.*—The two outstanding tissues were the mucosa and the bone, the most prominent pathologic changes being found in the former. This was more or less edematous and infiltrated with polymorphonuclear leucocytes or young connective tissue cells. The polymorphonuclear leucocytes occurred in clumps or individually, usually in large numbers. In cases of longer standing, chronic cases with acute exacerbations, the polymorphonuclear leucocytes were still in evidence, but the connective tissue cells were older and of the spindle shaped type. In certain specimens well established fibrosis was present. The bone was practically always sclerotic, with no evidence of bone spaces. About the only change present was some evidence of calcium absorption. Rarefaction, necrosis and hyperostosis were noticeably absent.

*Infant.*—I have divided these cases into two classes or types, according to the histopathologic picture:

*Class I, Edematous Type.*—The epithelium, which was stratified columnar in type, showed no more than two or three layers of cells, usually in the process of disintegration and slough-

ing; the cilia were not distinguishable. No basement membrane was seen. The tunica propria was much thickened and edematous, being more or less infiltrated with polymorphonuclear cells, singly or in clusters, young connective tissue cells in large numbers and red blood cells. (Fig. 10.) During a recent visit with Dr. Stacy R. Guild, head of the Department of Otological Research at Johns Hopkins Medical School, I had the pleasure of examining some beautiful serial sections of temporal bones removed from similar cases, in which the histopathology of the mucosa of the middle ear was identical with that found in these mastoid cells. (Fig. 11.) The periosteum was either normal or very slightly thickened. The bone changes, consisting of some fragmentation and varying degrees of absorption of calcium, suggestive of mild, very early necrosis, were so irregular that they could not be considered pathognomonic. The bone was, as a rule, cartilaginous in nature. (Fig. 12.) The interspaces, which normally contained a weblike areolar tissue, showed changes almost identical with those of the tunica propria, except that the polymorphonuclear leucocytes did not occur in such large numbers.

*Class II, Fibrotic Type.*—The epithelium showed a slightly more advanced stage of sloughing. The basement membrane was not recognizable. The tunica propria was the seat of the most marked and the most pathognomonic changes. The picture being a general fibrosis in the early or advanced stages, a more or less marked infiltration by polymorphonuclear leucocytes, eosinophiles, lymphocytes and red blood cells. (Fig. 13.) The periosteum consistently showed a more marked thickening than that found in Class I, except that there was more advanced fibrosis in the interspaces.

In the mucosa that was considered edematous there may have been a small amount of early fibrosis, probably an attempt to overcome the infectious processes. Likewise, some of the fibrotic specimens showed a few areas of edema. However, the edema unquestionably predominated in Class I as did the fibrosis in Class II.

True necrosis of the bone occurred infrequently. In certain specimens there was a marginal fragmentation and disintegration of the substance of the bone in which it showed

a ragged edge, thinned by absorption of calcium, but there was no evidence of a frank mass necrosis or osteoclastic activity as I have seen in bone removed from diseased sinuses. (Fig. 4.)

## SUMMARY.

1. The histopathologic changes found in the turbinates and the paranasal sinuses were as follows:

*Epithelium.*—Thickening; sloughing; polypoid degeneration; metaplasia.

*Basement Membrane.*—Thickening.

*Tunica Propria.*—Edema; polypoid degeneration; round cell and red blood cell infiltration; fibrosis; dilatation or compression of glands; thickening of blood vessel walls; engorgement of blood vessels.

*Periosteum.*—Thickening; cellular infiltration.

*Bone.*—Osteoblastic activity; osteoclastic activity; fibrosis; hyperostosis; osteomalacia; necrosis.

There were exceptions to the above findings, viz., that no metaplasia was found in the epithelium of the sinuses, and no goblet cells were seen in the mucosa of the maxillary antrums. The basement membrane was not usually distinguishable in the sinus mucosas.

2. A certain number of specimens from the mastoids of infants showing edema and marked cellular infiltration of the tunica propria sharply contrasted with others showing very little edema and marked fibrosis of the tunica propria. No pathognomonic bone changes were evident in either.

## CONCLUSIONS.

It is quite evident that the response of the mucosas of the nasal cavity, the paranasal sinuses and the mastoid cells and antrum to infection is similar in many respects.

Acute infection of the mucosa was characterized by the generalized invasion by young connective tissue cells, red blood cells and polymorphonuclear leucocytes, the edema of the tunica propria, the dilatation of the mucus glands and the absence of bone changes. Chronic infection was established by the thickening, the metaplasia or sloughing of the epithelium, the large numbers of older connective tissue cells in the tunica

propria, the absence of free red blood cells, the replacement of polymorphonuclear leucocytes by lymphocytes, the thickening of the blood vessel walls and the destructive changes in the bone.

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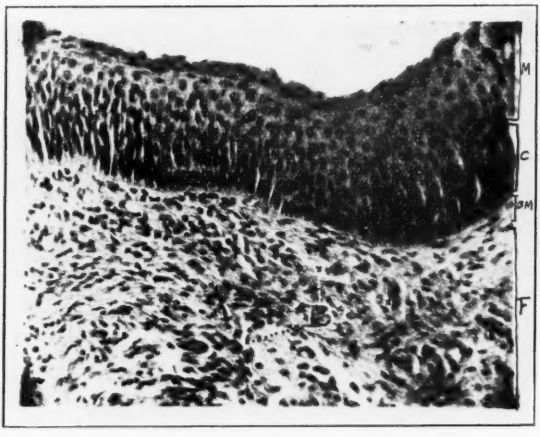



Fig. 1. Metaplasia of epithelium of turbinate with extreme fibrosis of tunica propria (× 500). M—Metaplastic cells. C—Columnar cells. F—Fibrous tissue of tunica propria. B—Blood capillaries. B. M.—Fibrotic basement membrane.

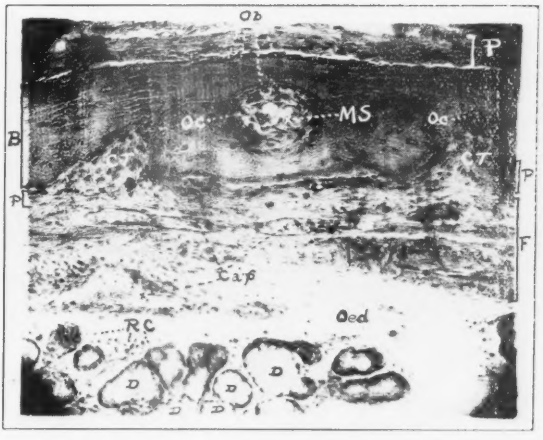



Fig. 2. Turbinate. Simultaneous osteoclastic and osteoblastic activity, with fibrosis of bone (× 300). P—Periosteum (extremely thickened). Ob—Osteoblasts. B—Bone. Ms—Medullary space. Oc—Osteoclastic activity from young connective tissue. CT—Connective tissue, following osteoclasts. Cap—Blood capillaries. Oed—Edematous tunica propria. RC—Round cell infiltration about dilated glands. D—Dilated glands. F—Fibrosis of tunica propria.

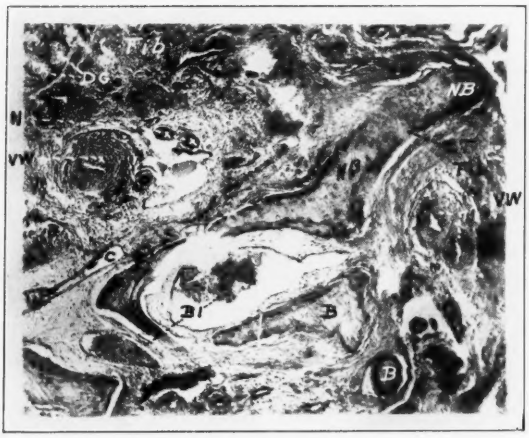



Fig. 3. Atrophic turbinate (× 175). Typical illustration of advanced generalized hyperplasia of turbinate. B—Bone. NB—Necrotic bone. CT—Connective tissue invading bone. VW—Blood vessel walls (extremely thickened). DG—Dilated glands. N—Nerves. C—Blood capillary (cut lengthwise). Bl—Free blood in blood space. Fib—Fibrosis.

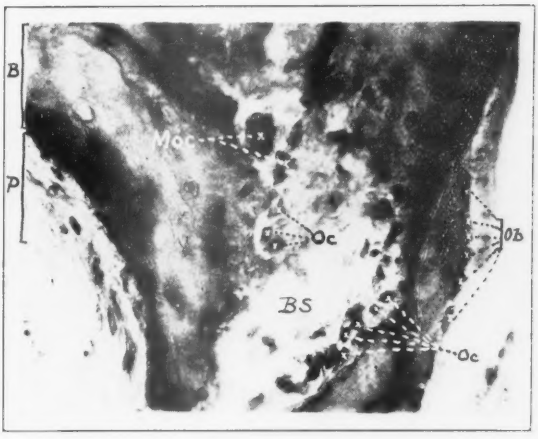



Fig. 4. Sphenoid wall (× 1000). Marked osteoclastic activity, with mild osteoblastic activity. Oc—Osteoclasts. MOc—Multinuclear osteoclasts. Ob—Osteoblasts. B—Bone. P—Periosteum thickened. Bs—Space from which bone has been destroyed.

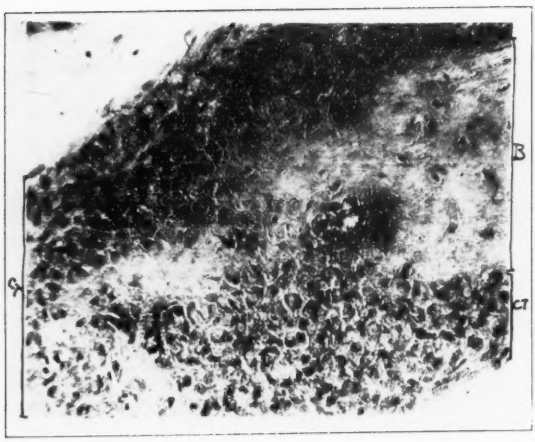



Fig. 5. Ethmoid wall (× 500). Young connective tissue cell invasion of normal bone; osteomalacia. B—Bone. CT—Young connective tissue cells.

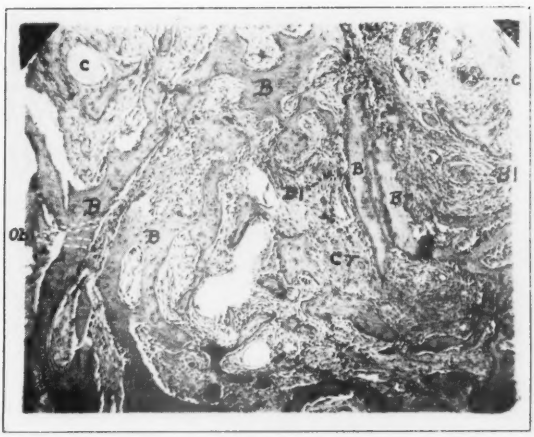



Fig. 6. Sphenoid sinus wall (× 100). Heteroplastic bone formation in young connective tissue. B—Bone. CT—Young connective tissue cells and fibrin. Ob—Osteoblasts. C—Blood capillary. Bl—Bone islands.

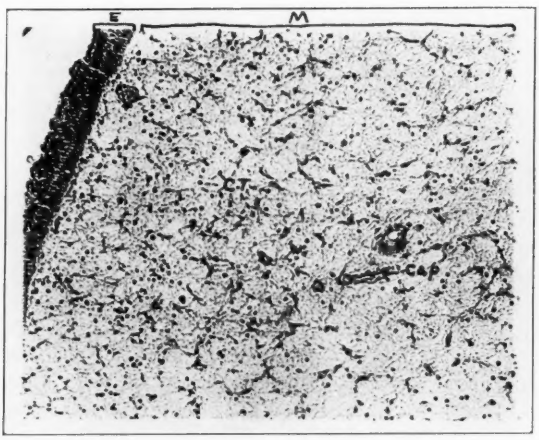



Fig. 7. Nasal polyp (× 135). E—Epithelium. M—Myxomatous tissue. CT—Connective tissue cells. Cap—Blood capillaries.

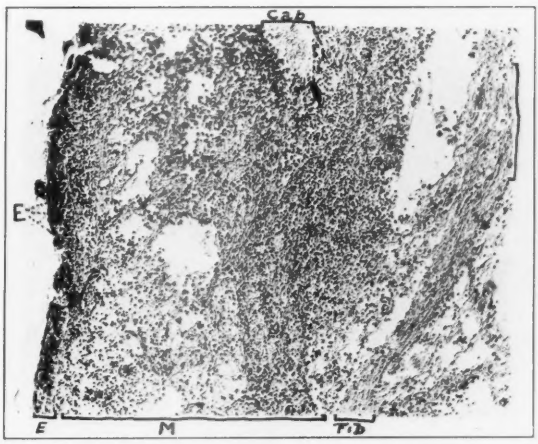



Fig. 8. Nasal polyp, removed three weeks after exposure to radium (× 135). E—Epithelium. M—Myxomatous tissue, densely infiltrated with young and old connective tissue cells. Cap—Dilated blood capillary. Fib—Fibrous tissue.

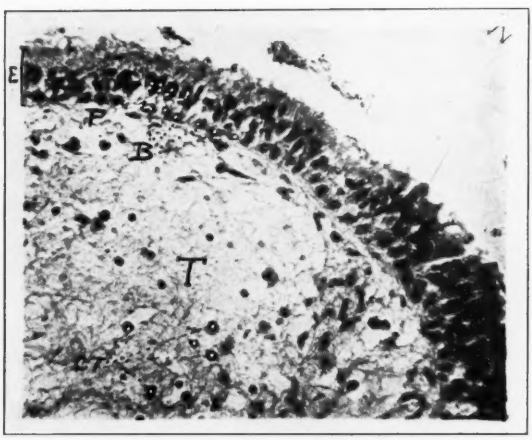



Fig. 9. Mucosa of frontal sinus (× 473). E—Stratified squamous epithelium thickened; cilia have sloughed. B—Basal cells. P—Pillar cells. T—Tunica propria, edematous; CT—Connective tissue cells.

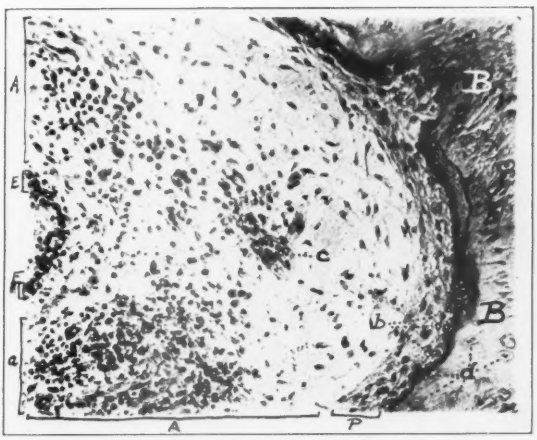



Fig. 10. Edema of mucosa in Class 1, infant mastoid (× 175). E—Epithelium sloughing. A—Edematous tunica propria. B—Bone. P—Periosteum. a—Young connective tissue cells and polymorphonuclears. b—Young connective tissue cells among which calcium is being laid down. c—Red blood cells and polymorphonuclears in blood capillary. d—Young bone cells.

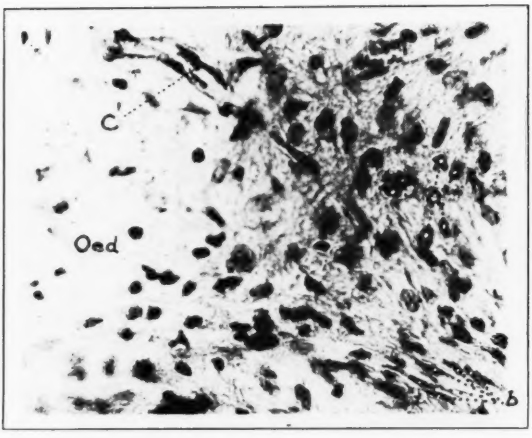



Fig. 11. Connective tissue cells in edematous tunica propria. Mucosa of infant mastoid (× 850). a—Young connective tissue cells. b—Older connective tissue cells. c—Blood capillary. Oed—Oedema.

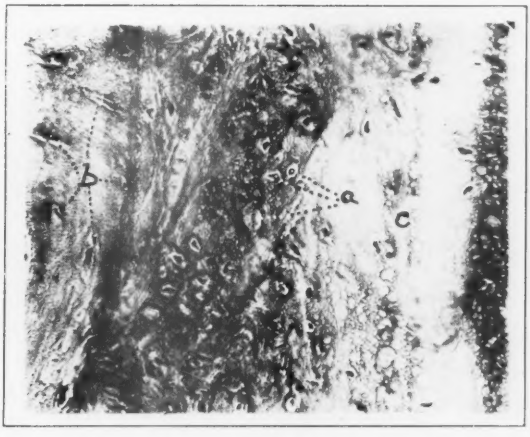



Fig. 12. Normal cartilaginous bone. Infant mastoid (× 500). a—Cartilage cells shrunken and stellate; some have dropped out of spaces. b—Young bone cells. c—Area from which calcium is absent.

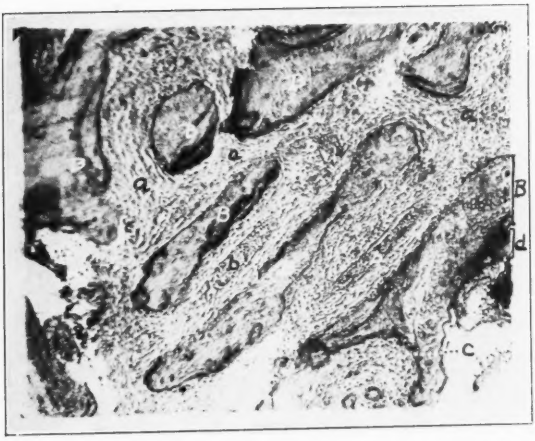



Fig. 13. Fibrosis of mucosa and interspaces in Class 2. Infant mastoid (× 135). a—Fibrous tissue, older connective tissue cells predominating. b—Blood capillaries, congested. B—Normal cartilaginous bone. c—Fragmentation of bone. d—Periosteum, thickened.

## LXIV.

# THE RELATION OF SINUS DISEASE TO HAY FEVER.

By T. E. Carmody, M. D., and L. W. Greene, M. D.,

Denver.

The literature available on this phase of the subject is very limited, and it appears that this most important condition has been very lightly treated; then, merely considered as unimportant, with the exception of Schadle, who was our pioneer in 1907, and some few men who have recognized the possibility that infected membranes in the various sinuses might be a contributive factor in hay fever. We take the stand that without infection in the sinuses or without pathologic membranes, our irritants, such as dusts, pollens, bacteria, foods and emanations of all sorts, etc., do not produce the socalled allergic coryza or hay fever.

We began our work on the subject by making the pollen tests in all cases. We used the pollen extract treatment with varying results, but in no case did we effect permanent relief. In many cases the condition was aggravated. One of us at that time complained of the classical symptoms of socalled "hay fever." Shrinkage of the nasal membranes alone did not give relief. A simple puncture of one of the maxillary sinuses, with irrigation, did give relief, however, from the symptoms which had been complained of for years before, although flakes only were recovered. One of us complained of these same symptoms, having been tested and found sensitive to the common ragweed. Simple puncture and washing of the maxillary sinuses relieved this condition. This gave us the incentive to find out if we could not relieve others so afflicted.

Our attention was drawn to a method which might prove more efficacious—i. e., we began a series by testing and X-raying all of our cases which showed clinical signs of "hay fever." The pictures proved in our first few cases that the membranes were involved in varying degrees.

In our large series of cases the patients presented themselves with the classical symptoms and history of exposure to the various irritants. Some had complained of these symptoms recurring each year, for periods varying from one to twenty years. These, in most instances, of course, were seasonal and due to the various pollens. In our locality the most common irritants are the ragweed group, the thistle group and the sage brush group. The clinical history in all cases gave the information that during the winter months they were comparatively free from symptoms. Schadle of St. Paul, in 1907, recognized the causative factor and attributed the infections of the antrum as the cause. Fortunately we were able to start our work by skin tests. In this series, due to the interest of the late Dr. Dean Beacom, associate professor of clinical diagnosis, University of Colorado, these cases were tested by him, then referred to us for clinical and X-ray check-up. In no case did the clinical and X-ray findings disagree, which is proof enough that pathology exists.

The next step was to see if these cases could be relieved. Treatment consisted of the usual shrinkage of the nasal membrane, etc. Many responded to the ichthyol treatment, others to simple puncture of the maxillary sinuses, and others (five in number) to radical operations. This work has been observed for the past three years without failure in any case.

We are all familiar with the various theories concerning "hay fever," namely, the toxin theory, the protein sensitization theory and the hereditary theory. Our concept deals with "membranes and filterable causative agents," which may or may not be a combination of the above theories or entirely independent.

The entire body, as we well know, is covered by a continuous protective enveloping membrane. This envelope, when not suffering from pathologic change or insult due to violence, is more or less impervious to outside influence. This phase of the subject does not take in the influence of toxins manufactured in the body itself and brought to the eliminating organs.

Death certainly, and possibly life itself, seems to be measurable by the parity of balance maintained between the factors which determine the nonentrance of outside elements, except food, oxygen and factors which assure perfection of elimina-

tion. A skin that is what we term "alive" is "waterproof," but the nearer it approaches a condition of dissolution, the more fluid or infection permeable it becomes. The paper thin intestine stands year after year as an almost miraculous barrier against infection, until some pathologic departure from normal renders it filterable to a toxic intestinal content.

The conjunctiva, although exposed through life to outside irritation and infection, remains, while normal and while bathed in that remarkable antiseptic, the tears, an impregnable defense; but let the continued absence of vitamins rob the conjunctiva of the elements necessary for its maintenance, it becomes an easily broken barrier with small protective properties. A nasal mucous membrane which is normal is (in consequence) resistant and nonfilterable to outside protein invasion, but it becomes, after greater or less period of abuse, due to faulty metabolism of protein from focal infection or faulty tissue chemistry, a spongy hypertrophied dropsical tissue, threaded with dilated capillaries, which give us our symptoms of socalled "hay fever." This membrane is now potentially a pathologic tissue and readily permeable to pollen proteins.

Most pathologic hypertrophies have back of them the very item which accounts for physiologic and natural increase in size and added amount of work. In a pathologic hypertrophy, the added work is called for because the tissue is compelled to do unnecessary and abnormal duty in the metabolism of faulty chemical substances brought to it.

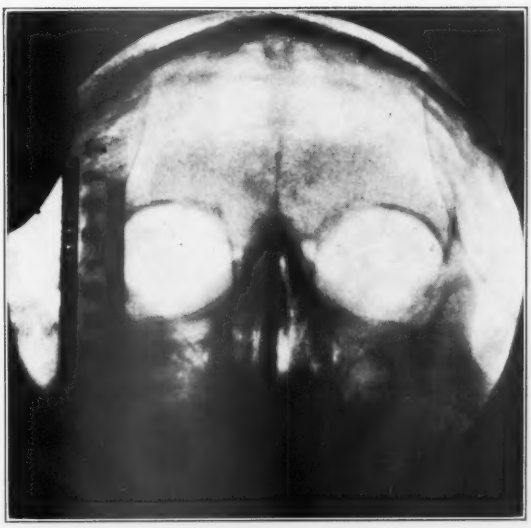
If we visualize the one great item of capillary caliber and physiologic blood vessel elasticity as a determiner of normal function, and classify a great majority of failures in anabolism and catabolism due to protein influence as "urticarias" of the tissue involved, many pathologic states will be rendered more easily understandable. A skin urticaria, a "big tonsil" or a large boggy turbinate, adenoids or generalized edematous nasal mucous membrane which allows a weed pollen protein to filter through it, a chronic or acute laryngitis, a bronchitis or asthma, are all departures from normal due to protein anaphylaxis, and all show the unstable dilated capillary. This may be accounted for by the biochemist with the acid combinations which compel engorgement of membranes, etc. Whether from sinus or other point of focal infection, such as food ingested, the results

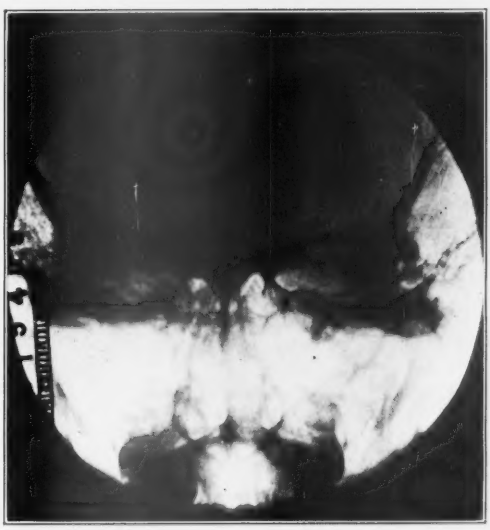
will practically be the same in any susceptible individual, namely, "local tissue dropsy."

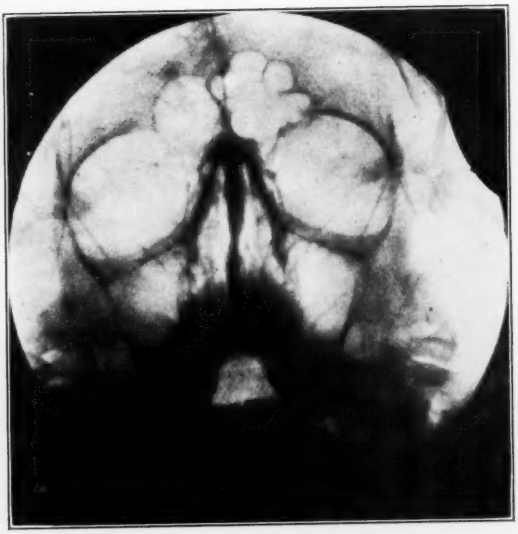
The cleaning up of a faulty body chemistry which has compelled an "urticarial" condition in given tissue, necessarily protects from further protein invasion through these tissues. In "hay fever" it would seem logical to ignore the weed pollen protein producing the distress, and instead to look to previous infection of, and keep the sinus mucous membranes in a state approaching normal and throw up a natural and physiologic barrier which cannot be broken down by extraneous invasion and "bombardment." No normal mucous membrane is filterable to an outside protein. If, in given cases, the emptying of an infected sinus and its subsequent healing brings about clinical cessation of socalled "hay fever" symptoms because the nasal tissues are no longer required to metabolize pus proteins which render them abnormal, would it not be a logical procedure to believe we are dealing with pathology of these membranes before an irritant comes along?

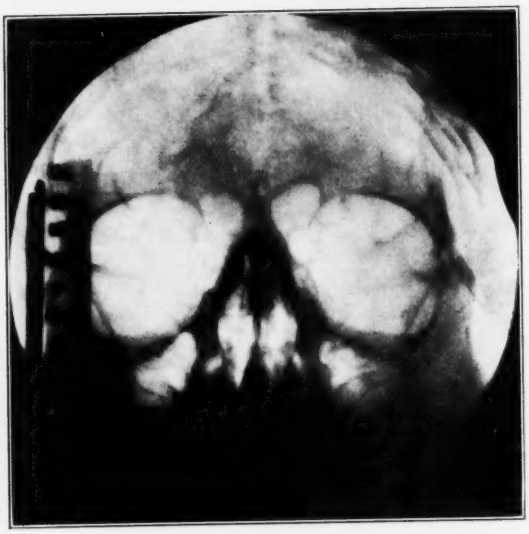
Treatment of these cases is divided into three heads: Those relieved by simple dehydration of nasal mucous membranes by the use of 20 per cent solution of ichthyol and glycerin tampons; those relieved by simple puncture of the maxillary sinuses; those relieved by radical operation on the sinuses. In our series, 77 responded to the simple ichthyol tampon treatment, 19 to the simple puncture of one or the other or both maxillary sinuses, and five to the radical operation on the maxillary sinuses and exenteration of the ethmoids.

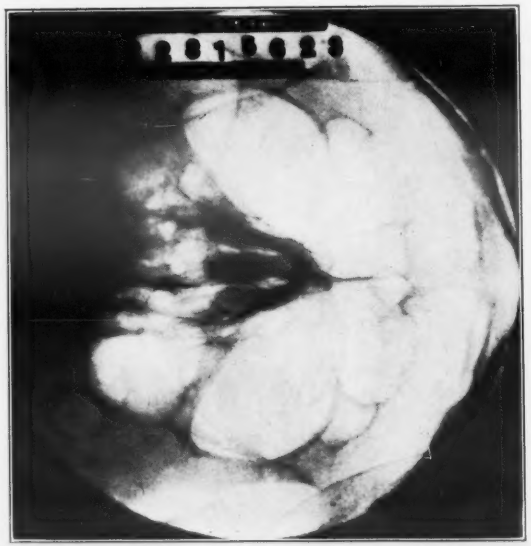
In summarizing, let us recall that we have dealt with but one phase of this vast subject, namely, "hay fever," as regards sinus infection, and we have found that the maxillary sinuses, as Schadle pointed out twenty years ago, are the most commonly infected. Next in frequency are the ethmoids, frontal sinuses and sphenoid sinuses.

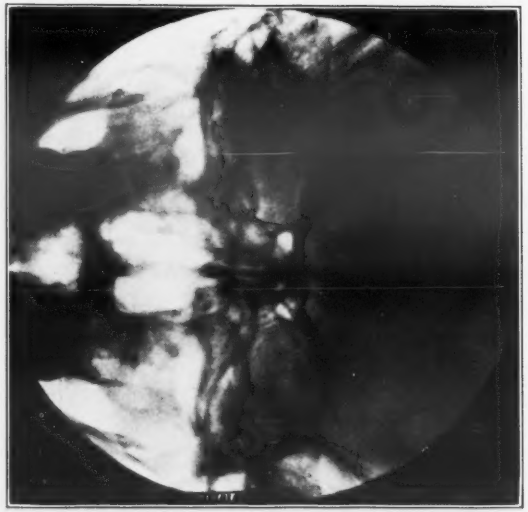












LXV.

# LIMITATIONS OF THE FUNCTIONAL TESTS IN THE DIFFERENTIAL DIAGNOSIS OF DEAFNESS.

By Edmund Prince Fowler,

New York.

The functional tests are employed to determine hearing capacity and to aid in differential diagnosis. These two objects are closely related, and we shall consider them largely together.

Any sound may be used to gauge the acuity of hearing, but all sounds are limited in various ways for functional testing. First, a sound must be loud enough to be heard. If it is not, it does not mean the patient cannot hear that particular kind of sound, but rather that it may not be loud enough for him to hear it. Consequently, to test the very deaf an intensity near the feeling point must often be employed to determine the degree of deafness. Most of the ticks and clicks of watches and coins and acumeters and the tones of tuning forks are deficient in intensity range and thus has arisen the belief that if one of them is not heard at its greatest intensity a tone gap exists.

One advantage of the audiometer is its great range in intensities. We have yet to note a tone gap in any of the thousands of cases examined, or any hearing for speech if none existed for the audiometer. Where other instruments have found tone gaps we suspect such an instrument of a too limited intensity.

The maximum intensities of my own forks, and the 1A audiometer are shown in Fig. 1 for both A. C. and B. C. You will note that whereas the audiometer gives great intensities by A. C., it is just as deficient in B. C. range (with the new B. C. receiver attachment), as are the tuning forks. It follows that if a patient's hearing is lowered beyond the capabilities of these instruments, it does not mean that he has lost all hearing for them but simply that we have not employed sufficient

intensity of vibration to determine his hearing. The intensity limits for B. C. by both forks and audiometer are much less than for A. C., and consequently B. C. may appear nil when A. C. is still measurable. It is unthinkable that with any residual hearing by A. C. there should not also be some B. C. and vice versa.

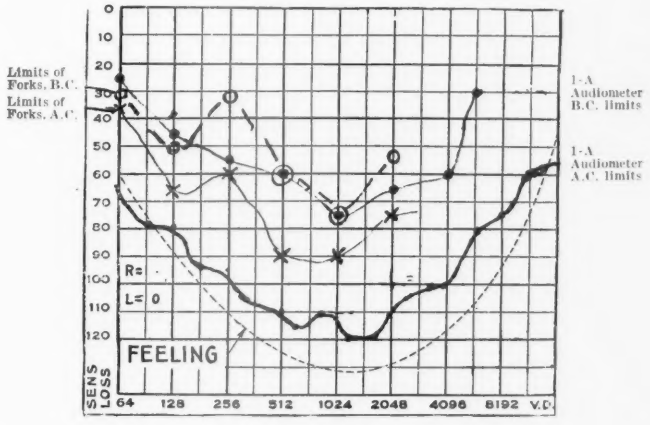



Fig. 1.

X—Limits of author's tuning forks by A. C.
●—Limits of 1-A audiometer for B. C.
○—Limits of author's tuning forks by B. C.

The voice is often used for testing the hearing. It is limited in several ways. For distances less than half a foot, results are very unreliable on account of reflections between tester and tested. For long distances reflections, standing waves and noises greatly interfere with accuracy. If there be insufficient space (there usually is) the intensity of the speech may be varied and the distance fixed.

*Calibrations of the voice in sensation units show that if a patient cannot hear the average soft whisper at 15″ his loss in hearing is greater than 30 S. U.; if he cannot hear the average

*Fletcher: Physical Measurements of Audition.

loud whisper (or softest voice) at 15″ his loss in hearing is greater than 45 S. U.; if he cannot hear the average medium voice at 15″ his loss in hearing is greater than 60 S. U.; if he cannot hear the average shouted voice at 15″ his loss in hearing is greater than 75 S. U.

The shouted word through a speaking tube is usable up to approximately a 100 S. U. loss. The voice used as above may be very useful to measure hearing for speech if great accuracy be not required. It does not aid much in differential diagnosis.

Speech is limited in another way. If it is increased gradually from minimum audibility to maximum loudness the ear is capable of understanding or interpreting the words spoken better and better, until the speech reaches a certain intensity (about that for the loud voice), when about 95 per cent are heard correctly. Then, as the intensity further increases, the ear hears and interprets the speech sounds less and less accurately.

At low intensities speech is limited either from lack of distance available or inability to produce very faint articulate sounds. All of these difficulties are overcome by the phonograph audiometer. The intensity of sound given off by the vocal cords when vibrating with the least possible amplitude (as with the softest voice) is only about 45 S. U. below the intensity of sound given off by the greatest amplitude of which they are capable (as with the shouted voice).

To be used as a gauge, a sound must be carefully measured either by comparison with a similar standard sound or by making it a standard by many tests of normal ears at its minimum audibility distance or intensity, but it has been quite the custom to assume that our whistle or our voice is similar in loudness and quality to some standard mentioned in some textbook and proceed accordingly.

Second.—The ear is so constructed that it is very inefficient at low and high frequencies, and very efficient in the middle frequencies, these latter being used most in hearing speech. A sound must lie within the range of frequencies hearable by the ear. If it does not, it matters nothing what intensity it possess, it cannot be heard.

The voices of man and woman vary in pitch, the latter being about one octave higher, and so it is stated that nerve or ob-

structive deafness is indicated if a deafened patient hears better male voices or female voices, respectively. Now, as a matter of fact, these are not heard better because one is pitched higher or lower, but because of differing intensity. The fundamental difference lies in the fact that man has twice the number of harmonies in his voice than has woman. He has twice the number because his fundamental is an octave lower and therefore his harmonies are spaced at half the distance apart, than are the harmonies of the woman's. It is by the spacing rather than by the fundamental frequency that the ear determines the pitch. The ear can even determine the fundamental of a sound, if the actual fundamental frequency is filtered out and no longer exists in the sound. In other words, the energy of woman's and man's voices varies as between people. The frequency content of both is the same, except in the low range, which does not count, because the human ear discriminates against low frequencies. Articulation tests have also indicated that practically the same results are obtained with the two types of voices. An exception to this is that obtained with the sibilant sounds which are more difficult to interpret when spoken by a woman. Male and female voices are limited by nature so that they cannot be efficiently used for the differential diagnosis of deafness, at least in the way taught.

The Galton whistle varies greatly in frequency and intensity, according to the air pressure exciting it, and also in accuracy of setting. Measurements show that the Edelmann-Galton whistle sound pressures persist well beyond the upper frequency limit of hearing. That is, though not heard, they may still register virtually the same pressures as when within the normal hearing range. The whistle emits at least two loud sounds, which vary not only with the force of the air blast employed but between instruments. The unaided ear can often differentiate several sounds. Which one the patient hears is sometimes a question. Even if it were a pure tone, unless it be calibrated for intensity it is purely qualitative (i. e., if it is not heard the ear is defective only for the intensities below that blown). If it is heard, the exact capacity of the ear to hear it is still impossible to determine unless we somehow predetermine intensity at different distances from the ear, and the loss indicated at such distances. This, if attempted, is found

to be impossible, because it is often heard better at greater distances than nearer. To demonstrate this standing wave (or maxima and minima) phenomenon with the Galton whistle move the whistle or the head slightly to and fro. Whenever testing for high tone perception similar movements should be executed to insure the maximum intensity reaching the ear. Some assume that if he hear the Galton note and his patient does not hear it, that the patient must have a nerve deafness. Let us both be tester and tested. Everyone in this room, please listen to the sound of my Galton whistle. I blow it as loud as is possible. Now, close your ears, preferably by pressing firmly against the right and left tragi with the tips of one of the fingers of your right and left hands, respectively. I again sound the Galton as before. Did you hear it? If you stopped up your ears sufficiently you did not. Have you nerve deafness now, but a moment before no nerve deafness? How about patients who do not hear it? Have they necessarily nerve deafness? They have not. It is easier to screen off high frequencies than low because the former cannot so easily leak around corners and through chinks. The loss of high frequencies may be caused by nerve deafness, obstructive deafness or both. The value of the Galton whistle lies in its ability to give great intensity. If heard it is certain that no total loss of hearing exists for the frequency used. If not heard, it may mean either nerve or obstructive lesion, or both, or neither.

The "lower tone limit" is elicited by a 16 D. V. to 26 D. V. fork. It is said to be raised if the ear does not hear these forks. Experiments show that at these frequencies measurements are very difficult to make, the thresholds of audibility and of feeling are often indefinite and vary considerably with observers, and the difficulty of exciting the fork to equal intensities, and the variations encountered in applying it make it impractical for use in measurements of absolute quantitative values. The same conclusions will be obtainable from using frequencies two or three octaves higher, so why make the lower tone limit tests? They are inaccurate quantitatively and not needed diagnostically.

The Rinne test as usually practiced is simply the difference between A. C. and B. C. We speak of its reactions as positive when A. C. is the normal number of seconds (whatever that

is) greater than B. C.; as diminished positive when A. C. and B. C. more closely coincide; as negative when B. C. is heard a greater number of seconds, or is heard longer than A. C., and as absolute negative when only B. C. is audible.

Rinne is limited in application, first, for the same reasons that all tuning forks are limited, namely, the intensities of forks are generally insufficient to produce a sensation of sound beyond limits as indicated in graph shown in Fig. 1. It may be negative, even absolute negative in undoubted nerve deafness, as shown in Fig. 2.

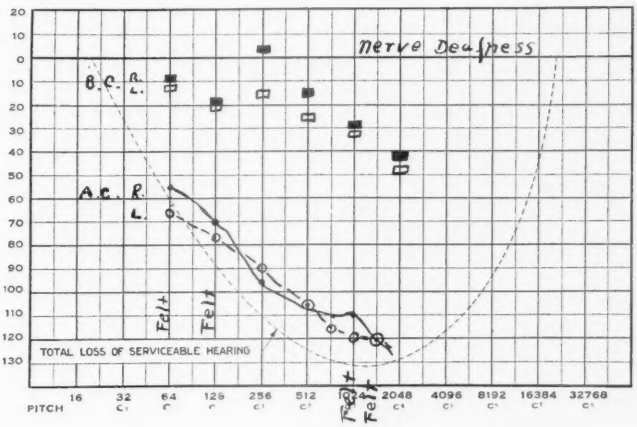



Fig. 2.

Second, by using the shank for the B. C. and the prongs for the A. C., we are really comparing two distinct instruments with different rates of decrement, and tuning forks differ greatly as to their relative differences between A. C. and B. C. This may be overcome, as I have shown, by the alternate method of placement, continued until no sound is heard by both air and bone conduction and by using calibrated forks so that the values by air and by bone will be in like units of measure. Also the new B. C. receiver attachment for the audiometer gives a rational method of determining B. C. and of comparing hearing capacity for both A. C. and B. C. in the same units

of measure. These, as shown, are deficient in B. C. intensity to measure great losses, but in so far as they go give what may be called a logical relationship between A. C. and B. C.

Third, many lesions may cause a loss in A. C. and no loss or even a gain in B. C. at certain frequencies (a Rinne negative), but we cannot tell to what extent the gain in one has offset the loss in the other, or vice versa. A markedly lowered B. C. with a gain in A. C., according to my records, never occurs. Experiments indicate* that middle ear lesions always cause unequal variations in A. C. and B. C., at some frequencies. This leads to the conclusion that no lesion producing like losses in A. C. and B. C. at the same frequencies can be a middle ear mechanical lesion and, if vestibulocochlea transmission be normal, it must be therefore largely nervous. It follows that in the average normal ear, if equal variations of A. C. and B. C. occur, these do not necessarily mean either nerve or obstructive disease, but it may be simply the normal variations for that normal ear. In an abnormal ear, if only equal variations occur, it means that either a nervous factor is operating or that the perception is lowered by some vestibulocochlea lesion. If unequal variations occur it means some mechanical factor is operating.

It is conceivable that some obstructive lesion occurring with a slight nerve deafness could greatly bring up B. C., and so confuse the picture that an erroneous diagnosis would be made. The losses possible by A. C. and B. C. from nerve deafness we know, because they may both be absolute. It is even more important to determine the variation limits of obstructive lesions by A. C. and B. C. When this has been done it will be possible at least to say "Only so much loss is possible by A. C., and only so much gain or loss is possible by B. C., and hence any surplus loss must be due to nerve deafness," etc. Before this society last year I tentatively proposed and followed such a plan in the differential diagnosis of deafness in school children.

Fourth.—A potent factor in diminishing B. C. is the damping of vibration by thick skull coverings; this can be discov-

*Fowler, E. P.: "Theoretical Interpretations of the Functional Tests." Archivees of Otology, 1927.

ered only by noting abnormal differences between two points upon the skull, and discrepancies as between the B. C. of the two sides of the skull, or any B. C. diminished proportionately more than is probable as compared with A. C. I have yet to note B. C. really diminished more proportionatley than A. C. It is seldom lowered as much, which strengthens the conclusion that the coincidence of A. C. and B. C. indicates no great mechanical cause for the deafness. A very large percentage of cases of deafness show a noncoincidence of A. C. and B. C. losses, and this would indicate that usually some mechanical factor is operating. Even this noncoincidence means little, unless it is certain that the opposite ear is not hearing the B. C. better than the ear being tested.

Fifth.—If Rinne is confined to one or even to a few frequencies it is unknown whether or not it be reversed at some of those not tested, because Rinne may hold true only for the frequency used. It may be positive at some frequencies and negative at others. In which case, does it mean that at the positive Rinne frequencies nerve deafness (or normal) is indicated and in the same ear at the negative Rinne frequencies an obstructive deafness, or does it mean that a large difference between A. C. and B. C. is needed to determine these alternativs? It would appear impossible to answer satisfactorily these questions, because the Rinne test is based upon a difference (not a ratio) between A. C. by one mode of application and B. C. by another mode of application. Moreover, it may be either positive or negative with forks of different frequency, and even with forks of the same frequency, because the decrement of different forks by A. C. and B. C. may vary to such an extent that the difference between the A. C. and the B. C. may be greatly changed, or even reversed in certain cases of deafness.

Sixth.—In unilateral deafness Rinne may be misleading because there is so little impedence to the transmission of sound from one mastoid to the other, that one ear may be even stone deaf and yet the vibrations of the fork upon it will be transmitted so efficiently to the opposite normal ear that no loss in B. C. may be noted, thus giving an absolute negative Rinne. Masking the good ear by a noise apparatus may cut out enough sound to aid us in this case, but if the better ear

is also notably down in hearing no noise apparatus yet devised will avail us, because deafness greatly lowers or annuls the masking effect. If hearing exists for B. C., hearing also exists for A. C., if the sound is made loud enough.

Instead of the Rinne test I prefer to compare absolute losses by both A. C. and B. C., in sensation units, at a spread of frequencies from 64 D. V., or 128 D. V., to 8000 D. V. or over. No complicated or uncertain measurements are then involved, and the relation of A. C. to B. C. is definitely determined in like units from the average normal. There is now no necessity for the Rinne test, and very little excuse.

The Weber test is regularly heard only in the ear to which it is lateralized, and so I believe Weber is due to a masking effect. Weber may be due to a lag (or acceleration) of the sound wave to one ear, more than to another, and (or) to the fact that the sound reaches one ear with more intensity. It appears that Weber is often referred to opposite ears by different frequencies. This may prove not a limitation, but a possibility for study, and such variations should not be ignored. Weber may be referred to one ear on light application of the fork, and to the opposite ear on firm application of the fork, or with the same application changed from one ear to the other as the fork dies down. Weber often cannot be employed in the very deaf because the fork may not be heard at all by B. C. It is dependable only when its findings can be correlated to the findings from other tests and clinical observations. It is generally unreliable and superfluous except possibly in monaural deafness.

The Schwabach test is limited as indicated in the discussion of Rinne because it too is heard by both ears, and unless the noise apparatus can mask it in the other ear we often cannot determine which ear is really hearing the sound. Schwabach is usually heard loudest in the ear tested. When patients hear the fork in the opposite ear, as frequently occurs, Schwabach is obviously not being measured in the ear of placement. Only in equal and similar binaural lesions, when both ears hear equally well by A. C., and in marked monaural deafness, it is serviceable for quantitative measurements.

Bone conduction sensitivity is not as accurately determinable as A. C. sensitivity, because so much depends upon the

technic of placement and pressure, and the reactions caused by the discomfort from these, and yet sometimes we hear quoted a diagnosis of nerve deafness made because B. C. is lowered a few seconds from the normal. Unless Schwabach shows a loss or a gain of at least 10 S. U. at several frequencies it means little. This is equivalent to twenty seconds with my 128 D. V. fork, using the alternate method. Many socalled prolonged Schwabachs only seem such, because the examiner's hearing is cut down by the masking effect of extraneous noise, whereas the deafened patient does not hear this noise.

Schwabach is limited in the frequency range available, because B. C. by the low forks is felt before heard, and at the higher frequencies it is audible through the air before the shank touches the ear; this may confuse the patient.

As all bone conducted sound reaches the ear largely, if not wholly, by way of the conducting mechanism, it is important not to limit B. C. to one frequency but to take a spread from 64 or 128 D. V. to 2048 or 4096 D. V. or higher. Fig. 3 shows how misleading it would have been to limit Schwabach to the lower frequencies, because it is normal or above at these and markedly down elsewhere. Fig. 4 shows B. C. normal and A. C. greatly diminished at all frequencies.

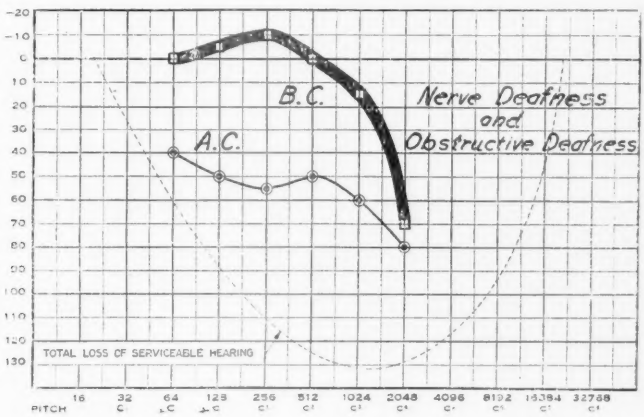



Fig. 3. Nerve deafness and obstructive deafness.

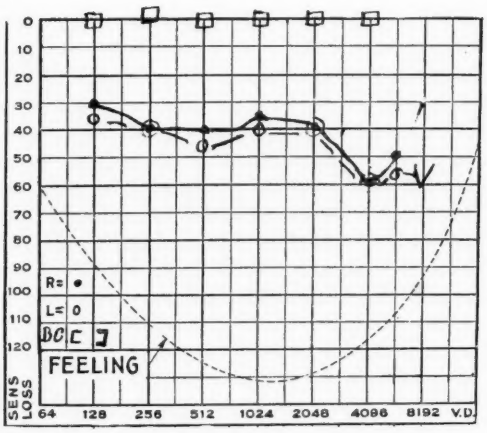



Fig. 4. Obstructive deafness.

The Gellé test, besides other uncertainties, is limited, like all B. C. tests, by possible lack of hearable intensity, by the patient's unawareness as to which ear hears the fork, by his mistaking changes of quality for changes in intensity and by faulty technic. The Gellé test, like the Weber test, may give exactly opposite findings at different frequencies. This may be due to the increase in tension of the membrana tympani, ossicles, etc., favoring the higher frequencies and greatly impeding the lower.

Schwabach, Rinne, Weber and Gellé all involve the use of bone conduction, either absolute or relative to A. C., and therefore are influenced by the same factors which limit the dependability and application of all B. C. data.

The noise apparatus of Bárány is very efficient for cutting out A. C. to the normal, or near normal, ear not being tested, but to be certain of its efficacy one should always first operate two simultaneously, one in the right and the other in the left ear. If no sound is then heard by the patient, remove the one in the ear to be tested and proceed with the test. If this technic is not followed, it is often uncertain in which ear the patient is really sensing the test sound. The noise apparatus is not

very loud in the mid and high frequency ranges, the ranges of speech and forks and Galton whistle. It is therefore deficient in the frequencies most useful for masking the test sounds usually employed. A noise apparatus should be adjustable in intensity, because a different volume of sound may be needed in different cases. Only sufficient noise should be employed to mask the test sound, as otherwise it will more markedly affect the hearing of the tested ear. Even with trained observers it will always affect somewhat both ears and thus tend to lower the hearing in the ear under test both by A. C. and B. C.

I have discussed mainly the physical imperfections and limitations of the functional tests, because unless these are kept in mind little accurate data will be obtained. The accurate measurement of A. C. and B. C., showing variations from the normal of both ears in like units of measure, appears to be the only logical procedure. The true interpretation of these variations will be evolved from correlating clinical and experimental observations and autopsy findings. The criteria at present relied upon for differential diagnosis, though seemingly qualitatively useful, may be very misleading, and often at variance with known experimental findings and acoustic laws.

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LXVI.

# ANATOMIC STUDIES OF THE SPHENOPALATINE GANGLION AND THE POSTERIOR PALATINE CANAL, WITH SPECIAL REFERENCE TO THE USE OF THE LATTER AS THE INJECTION ROUTE OF CHOICE.*

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Introduction.—In a study of the sphenopalatine ganglion and the various manifestations of its pathology, it is impressive to note the number of ailments that have been amenable to treatment by applications to or injection of this nerve center. While many rhinologists are daily successfully treating a great variety of painful or diseased conditions by injection of the ganglion, there are others who view with skepticism many of the results reported and are inclined to emphasize the technical difficulties of injection and the failures to obtain the desired effects. Some of these failures are, no doubt, due to improper injection of the fluid, others to variations in the anatomy in the region of the sphenomaxillary fossa, factors which could probably be largely eliminated by a more thorough knowledge of the variations which occur in the anatomy of these parts.

The injection of the ganglion can be made by three different routes: Through the nasal mucous membrane, through the pterygomaxillary fissure, and through the posterior palatine canal. The injection through the nasal mucous membrane was first used by Sluder and for years thereafter was the only means of approach. There are certain conditions, however, which make injecting by this route difficult, uncertain and sometimes dangerous. A marked deflection of the septum, a split septal cartilage, a large ridge or spur, a hypertrophied turbinate, or the presence of polyps may prevent the proper insertion of the needle. In some cases operations on the nose

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

have been performed obliterating the landmarks. Again, the nasal wall of the sphenomaxillary fossa may be so thick as to cause considerable difficulty in penetrating it. Then also there is the possibility of persistent bleeding, especially if the injection is made through the sphenopalatine foramen instead of under the posterior tip of the middle turbinate.

The method of injection through the pterygomaxillary fissure has been commonly used by dentists. It avoids the possible difficulties of the intranasal route, but there is still the uncertainty of exactly placing the solution in the ganglion.

The use of the posterior palatine canal as an injection route was first advocated, in 1921, by Carrea, a dental surgeon of Buenos Aires. He used this route for anesthetizing the maxillary nerve but did not mention the sphenopalatine ganglion. In 1925, when Ruskin first described the injection of the ganglion through the posterior palatine canal, a new impetus was given to the treatment of various neuralgic and other painful conditions by injection of the ganglion. After some experience with this method of injection, the writer found it possessed features of accessibility, accuracy and certainty of result not found in approaching the ganglion by either of the other routes. However, there were still present certain factors that made it at times difficult or at least uncertain. It was found, for example, that it was not always easy to locate the palatal orifice of the canal, as it varied somewhat in position and size. Sometimes the needle would traverse the soft palate or penetrate the tissues of the nose. Sometimes the needle would pass up the canal with but little resistance, while at other times considerable resistance would be met with, and in a few instances it would not pass at all. Again, the point of the needle would stop on the face of the sphenoid bone without having been inserted the necessary distance, or, on the other hand, it might not reach bone, no matter how far it was inserted. There was also the question of how far and to what structures fluid injected through this canal would spread and whether or not damage to the important nerves in that region might occur.

In the hope of clearing up some of these points and of developing a surer technic of injection of the ganglion the writer made some dissections from material available in the

University of Pennsylvania Medical School, and some craniometric measurements of skulls from the Hyrtl collection in the Mütter Museum of the College of Physicians of Philadelphia. The cranial study consisted in the examination of fifty skulls which belonged to people whose ages at their death ranged from thirteen to fifty years. They were all of white people living in central Europe, most of them in Austria. They formed a selective series, all skulls showing any gross abnormality and those from people who had died of any diseases causing a change in their skeletal framework being eliminated.

In this study it was hoped particularly that certain measurements could be made and certain indices established which would promote greater accuracy of result. In addition it was felt that a knowledge of the variations which might be found would not only explain failures in result but also point out ways of counteracting such variations. To this end the following measurements were made:

1. Distance from the posterior edge of the posterior palatine foramen to the anterior mesial edge of the upper middle incisor tooth on both sides.

2. Distance between the points of the cusps of the upper canine teeth.

3. Distance between the outer edges of the upper first premolar teeth.

4. Depth of the palate at the anterior edge of the first molar teeth. This measurement was obtained by passing a probe through a stiff piece of cardboard laid across the upper teeth so that the probe struck the center of the hard palate at the level of the anterior edge of the first molar teeth. Then this distance between the hard palate and the surface of the cardboard, representing the depth of the palate, was measured.

5. Distance between the outer edges of the two posterior palatine foramina.

6. Distance on both sides from the outer edge of the posterior palatine foramen to the inner lower border of the second molar tooth.

7. Distance on both sides from the outer edge of the posterior palatine foramen to the inner lower border of the second molar tooth.

8. Distance from the posterior edge of the posterior palatine foramen to the level of the lower edge of the sphenopalatine foramen on both sides.

9. Distance from the posterior edge of the posterior palatine foramen to the lower edge of the foramen rotundum.

10. Distance from the posterior edge of the posterior palatine foramen to the under surface of the lesser wing of the sphenoid on both sides.

THE POSTERIOR PALATINE CANAL.

1. In the first measurement—that is, the distance between the posterior edge of the posterior palatine foramen and the anterior mesial edge of the upper middle incisor tooth, it was found that the distance on the right side ranged from 4.8 cm. in the skull of the child, 13 years of age, to 6.4 cm., or an average of 5.46 cm. in the adult male skulls (those over 20 years of age), and an average of 5.28 cm. in the adult female skulls. On the left side the range of distance was the same, with an average of 5.79 cm. for the adult male skulls and an average of 5.28 cm. for the adult female skulls (five in number).

It was found that in some specimens the incisor teeth protruded forward sometimes rather prominently, so that a measurement of this distance gave a wrong impression of the length of the hard palate. Sometimes one middle incisor tooth would protrude and the other one be straight. These variations gave a difference in the distanec of the two sides of one to three millimeters. When the two sides were symmetrical, as far as the teeth were concerned, the distance was practically always the same on both sides, showing that the posterior palatine foramina were always in a symmetrical position.

2. The distance between the points of the canine teeth was found to range from 3.1 to 3.9 cm., or an average distance of 3.53 cm. in the adult male skulls and of 3.4 cm. in the adult female skulls.

3. The distance between the outer edges of the first premolar teeth was between 4.1 and 5 cm., or an average of 4.55 cm. in the adult male skulls and of 4.42 cm. in the adult female ones.

4. A rather wide variation was found in the depth of the palate, ranging from 1.4 to 2.6 cm., or an average of 2.23 cm. in the adult male skulls and of 1.8 cm. in the adult female skulls. In some specimens there was a rounded ridge running anteroposteriorly in the center of the hard palate, making the depth, as measured from the center of the hard palate, less than it would have been if measured further to either side of the center. In most of the specimens the greatest depth was from the center of the hard palate, the palate being in the shape of a smooth regular arch.

5. The distance between the outer edges of the two posterior palatine foramina ranged from 2.3 to 4.1 cm., or an average of 3.47 cm. for all the adult skulls. This measurement could be used as representative of the width of the palate posteriorly and was found to vary considerably. Some specimens showed an average width anteriorly as measured between the canine teeth but a posterior width that was as much as one cm. less or more than the average.

Comment.—A study of these five measurements fails to show any regular and constant relationship between the width or depth of the palate and its length. It was hoped this study might show that a certain width of the palate as measured between the canine or first premolar would mean a certain length of this palate so that a constant ratio could be established, but this was found not to be the case. For instance, in taking one specimen, we find the distance between the canine teeth to be 3.8 cm. and the distance from the posterior palatine foramen to the incisor teeth to be 6.2 cm., making a ratio of the width and length of 3.8 to 6.2. In another specimen we find the distance between the canine teeth to be the same (3.8 cm.), while the distance between the posterior palatine foramen and the incisor teeth was 5.4 cm., or a ratio of 3.8 to 5.4. The same variations were found in the width of the palate as measured from the outer margin of the first premolar teeth. A constant ratio between the depth of the palate and its length could also not be established. For instance, in one specimen a ratio of 2.2 to 6.2 was found, while in another specimen the ratio was 2.2 to 5.6.

It can be seen, therefore, that the exact location of the posterior palatine foramen cannot be determined by a measure-

ment of the width or depth of the palate. However, the study of these skulls has shown several anatomic features that make the location of this foramen rather easy, and a knowledge of these features will prevent the insertion of the injecting needle through the soft palate in most instances.

6. In none of the fifty skulls examined did I find the location of the posterior palatine foramen opposite the second molar tooth. In five of the specimens it was situated opposite a point about half way between the second and third molar teeth, and in thirty-six of the skulls it was opposite the third molar tooth. In nine cases it was found posterior to the level of the third molar tooth. Thus in 72 per cent of the specimens it was found opposite the third molar tooth. In practically every skull examined the foramen was situated close to the alveolar line so that a needle inserted close along the edge of the alveolus would follow along this edge and fall into the canal. In most cases the edge of the alveolus was rather rough, so that a needle point probably would not slide along it, but if the needle is kept within a millimeter or two of this alveolar line it will fall into the canal. The keeping of the needle close to the alveolar edge is also of importance in preventing the needle from occasionally slipping over the posterior edge of the hard palate through the tissues of the soft palate. It was found that the distance between the edge of the posterior palatine foramen and the edge of the hard palate varied from .1 to .5 cm. on both sides, or an average of .3 cm. on the right side and .33 on the left side. And it was found that this short distance was not directly posterior to the foramen but diagonally inward to it. Directly posterior to the foramen is the articulation of the pterygoid process to the hard palate. This forms an area of solid bone for a distance of 1 cm. posterior to the posterior palatine foramen. Therefore in inserting the needle, if it is kept close to the alveolar border it can go one centimeter posterior to the foramen before it goes through the soft palate, while if it is inserted a little further internally it may go through the soft palate as close as one millimeter from the posterior palatine foramen.

Another observation made was the fact that the angle the posterior palatine canal makes with the surface of the hard palate varies somewhat. It was found that when a probe was

inserted into the canal it came opposite the anterior border of the first molar tooth at the level of its lower surface, in some cases; in others, opposite the center of the first molar; and in still others, opposite the middle of the second molar. This variation is partly due to the difference in location of the foramen and partly due to the direction the canal takes. This observation is useful in attempting to insert a needle into the canal. In order to strike the foramen at the proper angle the needle must be inserted at a point where the shaft will be opposite the first or second molar tooth. Then, as it is pushed upward and backward, it will strike the hard palate opposite the third molar tooth and enter the canal in the majority of instances.

The opening of the posterior palatine canal through the hard palate in many cases was found to be rather funnel shaped, and in some cases, especially in the skulls of older people, quite large. In these cases a depression can be felt through the tissues of the hard palate corresponding to this funnel-like opening, and therefore it gives an easy means of finding the proper place to insert the needle.

Comment.—Probably then the best technic for inserting the needle in the posterior palatine canal is as follows: First palpate the hard palate in an attempt to find a depression corresponding to the location of the foramen. If this cannot be felt insert the needle first so that the shaft comes opposite the anterior edge of the first molar tooth close up to the alveolar edge, pressing the needle upward and slightly backward. If the needle does not fall into the canal, remove it and insert it again a millimeter or two directly posterior to the first insertion. In this way it may be necessary to insert the needle several times, but if the palate has been previously rubbed with 10 per cent cocain solution and painted with an antiseptic solution it causes no pain and gives very little chance for infection. When the molar teeth are absent the posterior edge of the hard palate, and especially the posterior end of the pterygoid process, should be palpated and the needle inserted first about two centimeters anterior to the edge of the pterygoid process.

7. The distance from the outer edge of the posterior palatine foramen to the inner lower edge of the second molar teeth

was found to vary from 1.8 to 3.3 cm., or an average of 2.55 cm. on the right side and from 1.5 to 3 cm., or an average of 2.65 cm. on the left side. This distance had no constant relation to the length, width or depth of the palate and was found to be of no value in helping to locate definitely the site of the foramen.

8. The measurement of the distance from the posterior palatine foramen to the lower edge of the sphenopalatine foramen was made to determine the average distance to the sphenopalatine ganglion. This measurement was obtained by inserting a probe into the posterior palatine canal until its point could be seen through the pterygomaxillary fissure at the level of the lower edge of the sphenopalatine foramen. Then a marking on the probe where it came opposite the posterior edge of the posterior palatine foramen was made and the probe withdrawn from the canal and the distance between the tip of the probe and the marking measured. The lower edge of the sphenopalatine foramen has been found to be on a level with the lower part of the ganglion. Therefore a measurement to the lower edge of the sphenopalatine foramen will also give the distance to the lower part of the ganglion. The size of the sphenopalatine foramen was found to vary greatly. In some cases it was as small as .3 cm. in diameter and in others as large as 1 cm. in diameter. And the size of this foramen did not vary according to the size of the skull, the age, or the sex. This also held true of the distance to the ganglion. This distance had no constant relationship to the size of the skull or to the length, depth or width of the palate. For instance, a large skull of a man with a long palate might give a shorter distance from the posterior palatine foramen to the ganglion than a small skull. However, in the majority of specimens it was found that the larger the skull the longer would be the distance to the ganglion and vice versa.

In this series of fifty skulls the measurement of this distance showed a variation of from 2.2 to 3.2 cm. on both sides, or an average of 2.85 cm. for the right side and an average of 2.58 cm. on the left side in the adult skulls. It was found in the dissection of fifteen specimens of sections of the face that the average thickness of the soft tissue over the hard palate was .77 cm. Therefore by adding this to the average depth of the

lower border of the ganglion from the posterior palatine foramen we find the average depth of the lower border of the ganglion from the mucous membrane surface of the hard palate to be 3.48 cm.

The descending branches of the ganglion have been found to come off directly from the lower border of the ganglion to pass down through the posterior palatine canals. Therefore, a needle inserted up through this canal will pass directly into the ganglion substance. It was found in these skulls that the tip of the probe inserted up the posterior palatine canal sometimes came close to the sphenopalatine foramen and at other times as far as 1 centimeter external to the plane of the foramen, showing the variation in the location of the ganglion in relation to the sphenopalatine foramen. This variation in location of the ganglion, as determined by the above method, also held true in an anteroposterior direction but to a much less extent.

It was found that in many of the specimens a probe inserted into the canal and directed externally as much as possible would pass out of the sphenomaxillary fossa into the zygomatic fossa and the point of the probe at the level of the sphenopalatine foramen might be as much as 2 cm. from the ganglion itself. In order then to have the needle pass directly up into the ganglion it should be inserted into the canal and directed internally to slide up along the inner wall of the canal. This inner wall extends up to the sphenopalatine foramen so that the needle directed along this wall cannot deviate from the right course. The external wall of the canal is of varying length, due to the variation in the extent of the articulating surface between the external pterygoid plate and the superior maxilla. In many cases the maxillary sinuses bulge posteriorly and this causes a wider articulating surface between the external pterygoid plate and the maxilla, and also tends to make the posterior palatine canal more narrow. In these specimens where the antra bulged posteriorly and there was a wide articulating surface the probe was found to fit sometimes very snugly in the canal and could not be deviated at all. In two specimens the posterior palatine canal was so narrow and probably irregular that there was difficulty in forcing the small probe up the canal.

In those specimens in which a shorter articulating surface between the external pterygoid plate and the maxilla existed there was a wider pterygomaxillary fissure. In these cases the probe inserted up the canal and directed externally would often go through the pterygomaxillary fissure out into the zygomatic fossa and strike the under surface of the greater wing of the sphenoid sometimes as far as 1.5 cm. from the border of the pterygomaxillary fissure. In some specimens the above condition was found to exist on one side but not on the other. In most specimens, however, the skulls were found to be fairly symmetrical as regards this region. In these specimens studied it was found that the probe could be passed out into the zygomatic fossa in fifteen of them, or in 30 per cent.

Of the fifty skulls the probe struck the anterior surface of the base of the external pterygoid plate thirty-five times, or an average of 70 per cent, when the probe was inserted straight in the canal. In some cases it was necessary to direct the probe posteriorly to strike this surface, and it was found that it struck the anterior surface of the base of the external pterygoid plate at the level of the middle of the sphenopalatine foramen twelve times, at the level of the upper edge of the sphenopalatine foramen fourteen times, at the level of the lower edge two times and at the upper edge of the foramen rotundum seven times. In some of these cases the probe would strike this surface and could not be directed any other place nor could it be forced beyond that level.

In those cases where the probe could be made to deviate more or less freely it was found that when directed externally it might pass out into the zygomatic fossa, when directed posteriorly it would strike the anterior surface of the base of the pterygoid process, when directed internally it would occasionally strike the upper edge of the sphenopalatine foramen but usually passed up along the inner edge of the foramen rotundum to go on up through the sphenomaxillary fissure to strike the under surface of the lesser wing of the sphenoid 1 to 4 millimeters external to the optic foramen. When directed anteriorly the probe would be apt to go about the same as when directed internally. Often the probe could be made to pass directly up in front of the foramen rotundum, usually by directing it internally as far as possible, but occasionally by directing

it more externally. It was found the probe could be directed up to the foramen rotundum in forty-one specimens, or 82 per cent. Occasionally this could be done only on one side. Therefore in many cases the injecting needle could be made to pierce the maxillary nerve as it emerges from the foramen rotundum.

9. In making measurements from the posterior palatine foramen to the lower edge of the foramen rotundum it was found that the distance ranged on both sides from 2.8 to 4 cm., or an average of 3.52 cm. on the right side and 3.49 cm. on the left side for the adult skulls. Thus with an average distance of 3.5 cm. to the maxillary nerve and an average distance of 2.71 cm. to the ganglion it would be necessary to insert the needle .79 cm. deeper than the level of the ganglion in order to have the injecting fluid have the most effect on the maxillary nerve. If it were possible to do so, directing the needle internally as far as possible would in most cases carry the point nearer to the maxillary nerve and in many cases directly into the nerve itself.

In most of the skulls examined (70 per cent) it was found that the probe could be passed on through the sphenomaxillary fissure up to the undersurface of the lesser wing of the sphenoid 1 to 4 millimeters external to the optic foramen. In some of these cases the probe could be passed only with difficulty, the point scraping along the bony walls. In three of the specimens the probe passed directly up to the lesser wing without touching bone elsewhere, and the probe could not be made to deviate from this course. In ten of the specimens the probe could be passed even further, on up through the sphenoid fissure to the top of the skull. In one case the probe passed directly up through this fissure without touching bone at any place.

10. In measuring the distance to the undersurface of the lesser wing from the posterior palatine foramen it was found that on the right side there was a variation of from 4.4 to 5.5 cm., or an average distance of 4.98 cm. for the adult skulls and on the left side a variation from 4.1 to 5.5 cm., or an average distance of 4.95 cm.

## THE SPHENOPALATINE GANGLION.

The remainder of these anatomic studies consisted in the dissection and examination of fifteen specimens. Each specimen consisted of half of a face, a mesial section having been made through the center of the skull, down through the center of the nose and palate. Each specimen was given an injection into the nasal ganglion region, through the posterior palatine canal, of 1 cc. of 70 per cent alcohol stained with methylene blue. The injections were made at depths of 3.5 to 4.5 cm. from the palatal surface: Seven at 3.5, 6 at 4 cm., 1 at 3.7 cm., and 1 at 4.5 cm.

The dissections consisted in the exposure of the ganglion, roots and branches by breaking through the lateral wall of the nose by means of rongeur forceps, and the exposure of the orbital tissues by removing the floor of the anterior cranial fossa. In this way the descending, ascending, internal and posterior branches, together with the maxillary nerve, its sphenopalatine branches, and the Vidian nerve were exposed, breaking through the sinus walls where necessary. Certain measurements were made of structures related to the location of the ganglion and the distribution of the injected colored fluid was carefully studied.

The various items to be reported on are as follows:

1. The thickness of the palatal tissue over the posterior palatine foramen. In this series of 15 specimens it was found that this thickness ranged from .5 to 1.1 cm. with an average of .77 cm.

2. Location of the point of the needle in the ganglion region. In some of the specimens the injecting needle passed up the canal very easily and could be moved laterally to some extent, but in others the needle fitted snugly in the canal and could not be deviated in any direction. In the former cases it was found that when the needle was directed externally the point would be at the outer edge of the ganglion, and when directed internally as far as possible it would be near the inner border of the ganglion. In these specimens the tissues in the ganglion were not all stained alike with the injecting fluid. For instance, if the needle was directed externally there would be only a slight stain on the internal branches of the ganglion. In the

cases where the needle could not be deviated from its course the point was usually found near the center of the ganglion. In nine of the fifteen specimens the needle point struck bone at distances ranging from 3.7 to 4.9 cm. from the palatal surface or an average of 4.22 cm. This bone proved to be the anterolateral part of the undersurface of the sphenoid sinus. This surface is rounded and it was found that in some of the specimens when the needle was forced it penetrated this thin bony wall into the sphenoid sinus, but in two of the cases the point slipped off the rounded surface and passed on up through the sphenomaxillary fissure.

3. Relation of the ganglion to the posterior tip of the middle turbinate. This relationship was found to have some variation. In most of the specimens the ganglion was situated just above the posterior end of the middle turbinate. In some cases the middle turbinate extended posteriorly beyond the location of the ganglion and in one case it extended as much as 3 cm. beyond the ganglion. In one case the ganglion was found opposite the posterior end of the superior turbinate. Another specimen showed the internal branches coming through the sphenopalatine foramen .5 cm. above and .5 cm. anterior to the posterior end of the middle turbinate. In these latter cases it can be seen that local application to the outer wall of the nasal cavity at the posterior end of the middle turbinate would not affect the ganglion.

4. Relation of the needle to the maxillary nerve. In the nine specimens where the needle could be passed beyond the sphenoid bone it passed in all cases but one just internal to the maxillary nerve as it emerged from the foramen rotundum. In this one case the needle point ran against bone .2 cm. external to the foramen rotundum.

5. Distance to the Vidian nerve. The Vidian nerve runs into the ganglion at its upper posterior border. It was found that this nerve, the internal and the posterior branches of the ganglion, come off at practically the same level. Therefore injections of fluid at this level would probably give the most even distribution to the different branches and roots of the ganglion. The Vidian and posterior branch in some cases seemed to be fused or bound closely together by sheath or fascia at their junction with the ganglion, but these nerve

trunks later separated to go through their individual canals in the lower wall of the sphenoid sinus. The needle point was found to be at the level of the Vidian nerve at a depth from the palatal surface ranging from 3.5 to 4.8 cm., or an average of 3.96 cm.

6. Distance to the maxillary nerve. The distance from the palatal surface to the level of the maxillary nerve was from 4.2 to 5.4 cm., or an average of 4.7 cm. Thus there was an average of .73 cm. between the levels of the maxillary nerve and the Vidian nerve. In addition to this, the maxillary nerve was found situated .5 to .8 cm. external to the Vidian nerve.

7. Location of the injected fluid when the needle was improperly inserted. In some of the specimens examined the inner wall of the posterior palatine canal at the level of the inferior meatus was found to be very thin, and in two cases there were dehiscences at this point, making an area about .5 cm. in diameter without bony support of the structures of the canal. Therefore it would be easily possible for a needle to penetrate this area and have the injecting fluid deposited in the inferior meatus. If the needle should be inserted back of the posterior palatine canal it might go through the soft palate directly into the nasopharynx, or it might pass up in the tissues of the anterolateral wall of the nasopharynx back of the posterior ends of the turbinates. The injected fluid then would be deposited either in the nasopharynx or in the posterior parts of the superior, middle or inferior meatus.

8. Distribution of the injected staining fluid. In all the specimens but one the staining fluid was found to have saturated the ganglion proper, but there was some variation in its spread to the branches and roots. In practically all cases the large descending branch was stained throughout its course through the canal. In all specimens but one the staining fluid involved the posterior branch and the Vidian nerve. In some cases the stain even passed through the Vidian canal throughout its length. In those cases injected at a depth of 3.5 cm. the stain did not extend up to involve the maxillary nerve except in one instance, and the ascending branches were stained only in their lower portion. In those specimens injected at 4 cm. the maxillary nerve was saturated in every instance as well as the ascending branches, the posterior branch,

the ganglion proper, the Vidian nerve and the descending branches. The internal branches escaped in some of these specimens, and in one case the upper part of the ganglion only was involved. In practically all of these specimens injected at a depth of 4 cm. the stain spread up the fascia around the ascending branches to reach the under surface of the orbital tissue, but the fluid did not penetrate into these tissues and did not come nearer than .75 cm. to the optic nerve or ophthalmic division of the fifth. In the specimen injected at 4.5 cm. the staining fluid saturated the maxillary nerve and extended to the undersurface of the orbital tissues but just touched the external surface of the ganglion, and no stain at all appeared on the internal and posterior branches or the Vidian nerve. Every specimen showed considerable stain in the tissues of the zygomatic fossa adjacent to the ganglion, the fluid reaching this region by oozing through the pterygomaxillary fissure.

In making these anatomic studies the writer was impressed by the variations in the anatomy of the sphenopalatine ganglion region and especially so by the differences in width, depth, etc., of the posterior palatine canal. Such variations as these can easily explain many of the failures to obtain the proper reactions following an injection of the sphenopalatine ganglion. It is so easily possible in many patients who have wide posterior palatine canals to direct the injecting needle some distance away from the ganglion so that the injected fluid would have no effect upon it. It is only by a complete knowledge of these many variations that the injection of the sphenopalatine ganglion through the posterior palatine canal can be successful in the greatest percentage of cases.

The writer wishes to express his thanks to Dr. George Fetterolf for the many valuable suggestions he gave in the preparation of this paper.

## SUMMARY.

1. In the hope of clearing up a number of uncertainties in respect to the injection of the sphenopalatine ganglion through the posterior palatine canal and in the hope of promoting greater accuracy of result, the writer made some dissections of the sphenopalatine ganglion and some craniometric measurements of fifty skulls.

2. A study of these measurements failed to show any regular and constant relationship between the width or depth of the palate and its length. A constant ratio between the depth of the palate and its length could also not be established. Therefore it was discovered that the exact location of the posterior palatine foramen could not be determined by a measurement of the width or depth of the palate.

3. The posterior palatine foramen was found opposite the third molar tooth in 72 per cent of the specimens, and in all instances close to the alveolar line.

4. After a study of measurements and variations in the anatomy of the hard palate a technic for locating the posterior palatine canal was decided upon as follows: First palpate the hard palate in an attempt to find the depression corresponding to the location of the foramen. If this cannot be felt insert the needle first so that the shaft comes opposite the anterior edge of the first molar tooth close up to the alveolar edge, pressing the needle upward and slightly backward. If the needle does not fall into the canal, remove it and insert it again a millimeter or two directly posterior to the first insertion. When the molar teeth are absent the posterior edge of the hard palate, and especially the posterior end of the pterygoid process, should be palpated and the needle inserted first about two centimeters anterior to the edge of the pterygoid process.

5. The measurement of the distance from the posterior palatine foramen to the lower edge of the sphenopalatine foramen which corresponds to the lower edge of the ganglion showed an average of 2.85 cm. for the right side and 2.58 cm. for the left side in the adult skulls. It was found in the dissections of fifteen specimens of sections of the face that the average thickness of the soft tissue over the hard palate was .77 cm. Therefore by adding this to the average depth of the lower border of the ganglion from the posterior palatine foramen the average distance from the lower border of the ganglion to the mucous membrane surface of the hard palate was found to be 3.48 cm.

6. Variations in the posterior palatine canal were found that would influence considerably the course of the injecting needle. It was discovered that in order to have the needle

pass directly to the ganglion it should be inserted and directed internally to slide up along the inner wall of the canal.

7. An average distance of 3.5 cm. from the posterior palatine foramen to the lower edge of the foramen rotundum was found. For the injecting fluid to have its fullest effect on the maxillary nerve the needle should therefore be inserted to a depth of 4.2 cm. from the mucous membrane surface of the palate.

8. The dissections consisted in the exposure of the ganglion, its roots and branches after an injection of 70 per cent alcohol stained with methylene blue had been made.

9. It was found that the location of the needle point in the ganglion region varied somewhat according to the amount the needle deviated from its course up the canal.

10. The relation of the ganglion to the posterior tip of the middle turbinate also varied considerably.

11. It was decided that injections of fluid at the level of the Vidian nerve would probably give the most even distribution to the ganglion, its branches and roots. The needle point was found to be at the level of the Vidian nerve at an average depth from the palatal surface of 3.96 cm.

12. In tracing the distribution of the injected staining fluid there was found to be some variation in its spread to the branches and roots, depending partly on the depth of injection and partly on whether or not the needle had deviated from its course up the canal.

## LXVII.

# CHRONIC NONPURULENT SINUSITIS AND ITS CLINICAL SIGNIFICANCE.*

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start with difficulties of terminology. In the literature of the subject we find the terms purulent sinusitis, nonpurulent sinusitis, catarrhal sinusitis, hyperplastic sinusitis, and others. Some of these terms are an attempt at a clinical classification and take cognizance of the diagnostic signs, while others are an effort to classify on a histologic basis. None of these terms are significant for the large group of cases with multiform combinations of any or all the qualities signified by the above terms.

Some rhinologists base their diagnosis of infection on the presence or absence of pus, and there seems to be a general impression among the profession at large that when pus disappears the infection is cured. I have several times watched the transition of an acute purulent antrum infection into a chronic catarrhal or hyperplastic type. Pus would be found only during exacerbations, but radiographs taken at intervals over a period of a year or eighteen months would show a progressive increase of pathology as evidenced by increase in thickness of the lining or the development of cysts or polyps. We have also had the experience of making a diagnosis of nonpurulent sinusitis, but on histologic examination of the tissues after operation found definite evidence of pus in the form of closed cystic abscesses or purulent infiltration.

We cannot draw sharp lines of demarcation in using the terms purulent and nonpurulent or hyperplastic and catarrhal

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In writing a paper of this kind we are confronted at the

sinusitis. We must recognize that one may merge into another and that there are all kinds of mixtures of type.

Several explanations might be offered to account for the different ways in which the tissues react to infection, as: difference in the biologic constitution, as seen in the allergic individual; differences in the infecting organisms; and difference in the stage of infection—acute, subacute and chronic, but this does not influence the fact that the absence of pus does not mean the absence of sinusitis.

In depending upon pus as an indication for treatment there is danger of overlooking the milder cases of sinusitis and the milder manifestations of coexisting infection in other sinuses. In consequence we may fail to cure the patient and may make it necessary for him to undergo a second operation. From a clinical standpoint it is often the nonsuppurative types that do the most damage. Neglect of this fact is one of the main reasons for the necessity of multiple sinus operations.

As in the clinical side so in the pathologic side there is no sharp transition from one type of inflammation to another.

Except in the extremes of type there is no outstanding difference in the gross appearance of tissues from chronic purulent and nonpurulent sinuses.

Histologic changes in the two stages of inflammation show a great similarity with only a few inconstant differences. Tissues from the suppurating sinuses are usually thinner and more vascular; those from the nonsuppurating sinuses will average a greater thickness; they also show more edema and a greater tendency to thickening of the basement membrane and to perivascular fibrosis with constriction of the blood vessels.

Cellular infiltration also is very similar, exceptions being that it is inclined to be more dense in the purulent cases and that eosinophiles are found more frequently and in greater numbers in the nonpurulent. As to changes in the epithelium, the purulent membranes may show areas where the epithelium is lost, sometimes with actual ulceration, while in the nonpurulent the change is more frequently to a hyperplasia or hyperplasia with mucoid degeneration.

Recurring repeatedly in the literature on sinus infections we find the statement that the epithelial layer is lost. This is not in conformity with our findings. In the majority of specimens we have found the epithelium intact. Even in the cases where the inflammation has been severe enough to cause ulceration or necrosis, we find large areas where the epithelium is still in place. This discrepancy in findings, we think, is due to lack of care in handling the tissues either at operation or in preparation for biopsy. The epithelium over the succulent tissues is very easily destroyed by such manipulations as sponging at operation or smoothing out before fixation.

That the epithelial layer does not offer a barrier to the passage of cells from the subepithelial layers is attested by the numerous sections in which the exudative cells are seen passing to the surface between the cells of the epithelium. We may have pus with an intact epithelial layer.

Anatomists tell us that in the mucous membranes of sinuses the basement membrane is absent or rudimentary. In the sections from sinuses with chronic infection it is nearly always present as a definite fibrous layer, frequently in a state of hyaline degeneration. The change is more pronounced in the nonpurulent than the purulent type. We look upon these changes as an evidence of chronicity, for the greater the chronicity the greater is the thickening of the basement membrane. None of these interstitial changes are constantly associated with either purulent or nonpurulent sinusitis. The only outstanding ones are the edema and the changes in the basement membrane as seen in nonpurulent sinusitis.

A comparison of the bacterial flora from different stages of sinus infection has been interesting. We have routinely taken material for cultures from two, and, when possible, from three sources at the time of operation. When the sinus is opened, swabs are taken from its contents or brushed over the lining. If cysts are present, their contents are aspirated, then the tissues removed are cultured after being washed and ground in sand. The growths from these cultures, from all types of inflammation, were, in the order of their frequency and predominance, streptococcus, staphylococcus, bacillus Friedlander, micrococcus catarrhalis and bacillus influenzæ. There was very little difference in the growths from swabs and

tissues except that the swabs showed more mixtures. From the cases without pus streptococcus was the predominating growth in 94 per cent and was present as a definite growth in 96 per cent. Of the cases with pus, staphylococcus was practically always present, but it was found in pure culture only in cases of acute empyema. Cultures from the contents of serous cysts were positive for streptococcus in 46 per cent; a few were mixed with staphylococcus.

Broadly speaking, the streptococcus is not a pus producing organism. The outstanding feature of streptococcic infection is edema, and these infections are prone to carry on in a subacute and chronic form. Staphylococci are pus producing organisms; these infections are acute and self-limited or spread as an acute process.

The most uniform interstitial change found in the tissues from chronic nonsuppurative sinuses is edema, and thickening or hyaline degeneration of the basement membrane, and, as stated above, they practically all cultured streptococci.

Admitting that some chronic sinuses are suppurative or purulent, it is my experience that there are more chronic sinus infections without pus than with it. I believe that nonpurulent sinusitis is the usual latent or chronic phase of sinus infection. This view is supported in a measure by the histories of the cases and by the bacteriologic and histologic findings.

Diagnosis is sometimes extremely difficult, particularly in the cases where structural changes are slight, but it is important to recognize these slight changes because I am sure that the seriousness of the sequelæ of sinus infection is not in exact proportion to the amount of pathologic change in the tissues. Radiography has been indispensable to us in the diagnosis of these latent cases.

Cysts and polyps are the result of infection. When they are seen intrasinus in the radiographs the diagnosis is made, but they may be present and not revealed by radiography.

Extreme infiltration or massive edema usually produces a blurring of the outline of the sinus or a thickening of the borders with an irregular outline raised from the bony margins. Dense infiltration or fibrosis produces a uniform increase in density. This is often interpreted as a sinus filled with pus. Lavage, however, may give a negative result.

When the structural changes are slight or the evidence of polyp or cyst is only presumptive, the injection or suffusion of contrast media, lipiodol preferably, has been an invaluable aid. Any increase in thickness of the lining over one-half millimeter we consider suspicious.

The object of this paper would not be advanced by going into the more obvious signs of chronic sinusitis as seen in the nose, but I want to record that of seven cases of choanal polyps and one case of choanal cyst upon which we have done radical sinus surgery, all but one, a polyp, had their origin from the lining of the antrum on the external lateral and posterior wall. None were attached near the osteum.

In the case of the antrum much may be learned from the cytologic examination described by Dr. Sewall of San Francisco. The technic, in brief, is to introduce into the antrum, through the ordinary puncture needle, five to ten cc. of sterile distilled water and reaspirate as much as possible for examination. The antrum should then be washed with warm saline solution to free it from the irritating distilled water and to gain what information you can from lavage. The aspirated fluid is examined macroscopically. It not infrequently shows gross masses of mucus, shreds and flakes, when the lavage will be clear. This fluid is then centrifuged and the sediment examined under the microscope. The sediment may show many leucocytes, lymphoid or plasma cells from a fluid that appeared perfectly clear. Gross particles may contain pus cells but are frequently made up of epithelial cells and cell débris held together by mucus. The entire mass of a very dense sediment is often made up entirely of débris and epithelial cells in various stages of disintegration. The liberated nuclei of epithelial cells may be, and I think frequently are, mistaken for round cells; however, massive sediments of this type are as significant in the diagnosis of chronic infection as the finding of many exudative cells. If one insists upon the presence of a certain number of true exudative cells in his cytologic examination he will miss many cases of chronic sinusitis.

Histologic examination of the lining of these antrums explains why there is so much of this débris and exfoliated epithelium. The subepithelial tissue will be edematous, with a moderate degree of cell infiltration. The epithelium will be

in a state of hyperplasia and metaplasia. There will be several layers of columnar cells with the surface layer in a state of mucoid degeneration, with exfoliated epithelial cells in the surface exudate. In some sections it will be hard to find a cell that has not become a goblet cell.

I think that in this condition these cells are cast off very rapidly because I have frequently found the field full of columnar epithelial cells with actively motile cilia. Ciliary activity will not be seen, however, unless normal saline solution is used in doing the cytologic examination.

When the attention of the profession was first attracted to foci of infection as a cause of systemic disease, infected tonsils and dental sepsis were considered the most important sources. Nasal sinus infection was relegated to a secondary place and attention paid most particularly to the more florid forms. The reason for this was that these infections were more obvious, and were more easily eradicated than that of obscure sinus infection.

From the clinical standpoint, practically any disease which can be produced by focal infection anywhere can be produced by sinusitis. We will consider a few that demonstrate the importance of latent infection and show its influence in some of the more obscure systemic disturbances.

The relation of sinus infection to bronchitis and asthma has been discussed so much that we mention it only to cite a few clinical coincidences in our experience. Bronchitis with copious purulent expectoration has usually been associated with purulent sinusitis. Bronchitis with an intractable cough that is productive of only a small amount of serous or mucus expectoration is associated most frequently with nonpurulent sinusitis. This cough is much like that from pressure, as with a substernal goiter, but the cough from pressure is usually nonproductive. Results following the cure of sinus infection in chronic sinus bronchitis have been the most satisfactory of any in our experience.

Most of our asthmatics have had sinusitis, usually of the nonsuppurative type. It is rarely confined to one sinus, and the majority of our asthmatics have had pansinusitis. Results from operation have been variable.

The diseases of the rheumatic group are essentially self-limited, but among those that have a tendency to persist, in a subacute or chronic form, we have found many with a latent or nonpurulent sinusitis that had been overlooked or considered of no consequence in the search for foci of infection, as in the following case:

Man, age 43, suffering from a subacute polyarthritis for nine months. No dental sepsis. Tonsils had been removed without benefit. Evidence of other sources of infection lacking. Sinus diagnosis was inconclusive, but there was evidence casting suspicion on the antrums. It was explained to the patient that we were unable to make a positive diagnosis of sinus infection, but that we advised exploration of his antrum. To this the patient readily agreed. At operation the lining of the right antrum showed a slight increase in thickness with two small glandular cysts in the alveolar recess. The lining of the left antrum showed a moderate uniform thickening throughout. Both membranes cultured streptococci that had a specificity for the joints of rabbits. Before operation this patient required assistance in dressing, and when walking went with two canes. Three days after operation he could get from bed and into his dressing gown with ease and comfort. He made steady progress and now, with orthopedic correction of secondary structural changes in his feet and ankles, he goes as a normal man.

Chorea minor has long been accepted as a disease due to focal infection. Like rheumatic fever it runs a definite course. But some cases go on to chorea intermittens, or even chorea permanens. We have had five cases of this type. The duration was eighteen months to three years; ages, 9 to 13. All had had tonsils and adenoids removed with temporary or no improvement. All had multiple chronic sinus infection. Two had purulent antrums. Two had latent infection in both antrums without purulence. One had purulent antrums with a nonpurulent ethmoid and sphenoid on one side. All showed immediate and definite improvement following exenteration of the antrums. The case with ethmoid and sphenoid infection, however, relapsed and did not show a permanent improvement until the ethmoids and sphenoids were operated upon.

Sinus infection has been a factor in a number of troublesome disease condition not so well defined, as in the following cases:

1. Secondary anemia with chronic cholecystitis and chronic nonpurulent sinusitis. The anemia persisted in spite of the usual methods of treatment. A double radical antrum operation was done. Following this, with no other change in treatment the blood picture rapidly returned to normal.

2. Tachycardia with dyspnea and fatigue, of unknown origin. Patient entered the hospital complaining of fatigue, dyspnea and rapid heart action. Examination showed mild diabetes, chronic cholecystitis and tachycardia up to 110. Detailed cardiovascular examination gave essentially normal findings. Accurate control of the diabetes did not relieve the fatigue and dyspnea. Following operation for chronic nonpurulent sinusitis all of the subjective symptoms were entirely relieved. Acute infections do disturb the tolerance of the diabetic, but in our experience the cure of chronic infection in a large number of diabetics has not shown that it had any beneficial influence on the diabetes itself.

3. Infectious atheromatous type of cardiovascular disease with cardiac failure. The overcoming of the cardiac failure was not possible under control. Septic tonsils were removed without result. On the radical removal of the antrum disease recompensation of the heart took place promptly. The patient gained a greater degree of health, strength and absence of dyspnea on exertion than he had had for two years previously. He resumed his business and indulged in moderate outdoor sports.

Recently we have been very much interested in a group of tic cases in which we found chronic sinus infection; the possibility of foci of infection being an etiologic or contributory factor in these disturbances having been brought to our attention by Dr. Laurence Selling. So far we have found definite foci of chronic infection in every tic case we have examined. Following is a brief synopsis of the outstanding facts from the histories of the three cases that have been longest under observation:

Boy, age 14, had his first appearance of tic following a prolonged sinus infection at the age of 6. From that time until

his sinus operation there were recurrent sinus infections, always accompanied by exacerbations of the tic. Gradual lessening in the frequency and intensity of the infections was accompanied by an improvement in the general physical condition and a lessening of the tics. Some months before his operation he had a subacute flare-up of his sinus infection, accompanied by a violent recrudescence of his tics. His early sinus infections were purulent. During the last flare-up no pus was seen (radiographs indefinite, showing only a suspicion of thickening). Three antrum punctures at weekly intervals showed no macroscopic pus, but always several polymorphonuclear cells to the field in the cytologic examination. At bilateral radical antrum operation we found in both antrums a moderate soft uniform thickening without suface discharge. It is nearly a year since his operation. He still has an occasional blink or twitch. To the casual observer he is a normal but slightly nervous boy.

Boy, age 11. Tics began insidiously between age of 6 and 8. Became intermittently more pronounced until age 10, when it was difficult for him to remain in school on account of his explosive speech and jerking and twisting. In his eleventh year he had violent attacks, during which he became uncontrollable. His sinus history was negative. Tonsils were out. Antrum lavage and cytologic examination were negative, but there was sufficient radiographic evidence to justify exploration of the antrums. At operation the right antrum showed a moderate thickening and edema throughout and the lining contained three glandular cysts. The left antrum showed a definite hyperplasia in the alveolar recess. The rest of the lining showed very little change. Following the operation there was a striking improvement, but when he returned to school there were several other severe recurrences over a period of three months. During the last quarter he has remained a quiet, contained and almost normal boy.

E. R., boy, age 14. Tics began with an acute onset, without apparent cause six months before our examination. Nose and throat diagnosis was infected tonsils, purulent antrums and a suspicion of infection in the ethmoids. Tonsils and adenoids were removed with no improvement following. Bilateral radical antrum operation showed a definite thickening with poly-

poid edema and some fibrosis. Following the antrum operation there was a definite reduction in the frequency and intensity of the movements, but at the end of two months the improvement not having come up to our expectations we exenterated the ethmoids and sphenoids. The lining of these cells was definitely hyperplastic throughout. Since this operation there has been a slow but steady improvement. He has slight residual tic movements, but is almost back to normal.

The first of these cases was under my observation for eight years, during which time there was a transition from a suppurating sinusitis into a latent chronic sinus infection without purulency and with only moderate changes in the sinus lining. The second had nothing in his history to suggest sinus infection, which was found only after careful examination of his sinuses. The third had multiple infections but did not recover until treatment had been carried to the limit of his least obvious infection.

I am firmly convinced that the infection in chronic sinuses is as great if not a greater source of trouble than the chronic tonsils, and that our failure to recognize it is responsible for our failure to relieve many of our patients suffering from focal infection. Another cause of failure is conservatism where only radical measures can eradicate all of the infected tissues. We have seen a number of instances where intranasal drainage of suppurating antrums put an end to purulency, but where relief from the secondary disease was accomplished only after radical exenteration. I believe it is just as important to remove the entire mucoperiosteal lining of an infected sinus as it is to remove the whole tonsil.

I think that purulency depends upon the type of infecting organism and the stage of its activity and that pathogenic organisms capable of doing serious damage may be harbored in a sinus without producing surface discharge but always causing structural changes in the tissues of the sinuses.

Diagnosis should be based upon the evidence for infection, whether surface discharge be present or not.

## LXVIII.

# THE USE OF THE NEW MAGNESIUM ALLOY TUNING FORKS.*

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Chicago.

For the functional testing of hearing, many methods have been devised. Although the human voice is difficult of standardization as regards intensity and pitch, still in many ways it is the most practical method. If care is taken to use only the residual air for unaccentuated conversation or whispered voice, and every effort made to employ the same intensity and pitch, with the patients always standing in the same part of the room, the eyes closed or averted from the speaker, with no part of the body touching the walls or furniture, using both high and low pitched tones, approaching the patient from the greatest possible distance, and noting at which point he first begins to answer properly; if these various precautions are observed, sufficiently definite conclusions may be reached.

After all, the actual efficiency of the ear is its ability to understand the spoken voice and, therefore, even though the tests cannot be made entirely accurate, they are very valuable from a practical standpoint. If the patient is able to hear sounds below one hundred double vibrations or above 4,000 double vibrations, but is unable to hear those between 100 and 4,000, he is deaf to all intents and purposes because he cannot understand the spoken voice. While the fundamental sounds of speech lie in the range between b-1 and g-2 (according to the theory of Bezold), we must remember that the overtones or harmonics which give a special quality to the voice have a much higher range, the overtones of some of the vowels such as e reaching above 3,000 double vibrations.

The watch, acoumeter and other instruments have been used, but these, from a scientific standpoint, are not very valu-

*Read before the American Otological Society at Atlantic City, May 24, 1929.

able. It is upon the tuning forks that we have learned to depend in a very large measure for functional testing. In recent times audiometers of various types have been added to our instrumentarium.

I believe that some of the requisites of good forks are these:

First.—They should be made of one piece of metal.

Second.—The handle should be long enough so that it can be easily yet loosely held without damping or stopping the vibrations.

Third.—The forks should sound for a long time.

Fourth—There should be weights on the lower pitched forks to avoid overtones.

Fifth.—Forks should not be nickeled.

Sixth.—If rustless metal is feasible, it should be used to prevent changes by oxidation.

The best forks have usually been unnickeled, but unless great care was used in handling them, and in protecting them from the moisture in the atmosphere, rusting took place and this changed the pitch. On the other hand, when forks are nickeled, the plating sooner or later begins to peel, and the vibrations of the loose pieces of metal give rise to adventitious sounds. For this reason, the Committee on the Standardization of Tuning Forks and Hearing Tests of the American Academy of Ophthalmology and Otolaryngology (of which I have had the honor of being chairman since it was formed in 1921), has been endeavoring among other things to find a metal or alloy which is rustproof and at the same time has sufficient elasticity so that it can be used in the making of first-class forks. A number of metals and alloys has been investigated, but none of them seemed to possess the proper qualifications. Finally, Mr. B. E. Eisenhour of the Riverbank Laboratory (founded by Col. Fabyan), at Geneva, Illinois, obtained a magnesium alloy consisting of 95.6 per cent magnesium, one of the lightest metals known (with a specific gravity of 1.7), 0.4 per cent manganese (specific gravity 8), and 4 per cent aluminum, the latter having a specific gravity of 2.7. This alloy, therefore, has a specific gravity of about 2.2; in other words, one-third that of steel, which is usually 7.7. The two great advantages of this alloy are that it is rustproof and very light in weight. After a time the alloy changes color but does

not peel. When using the low pitched heavy steel forks, the fingers tire quite easily after making a few tests. With the magnesium alloy forks, this difficulty is eliminated. A complete range of forks extending from C-2 (16 double vibrations) to c-5 (4,096 double vibrations) has been perfected by Mr. Eisenhour. The price of these forks is about 25 per cent less than the Edelmann series. With ordinary careful manipulation, these forks do not seem to break or become damaged any more easily than do the steel ones.

The "constant" of damping or decrement, the use of which was suggested by Harvey Fletcher for purposes of comparison with other methods, can be supplied for each of these forks at a cost of $1.50. By employing Fletcher's formula, one may determine the loss of hearing figured in sensation units just as is done with the audiometer. Knowing the length of time any particular fork is heard by the normal individual, subtracting from this the time that the fork is heard by the patient, and multiplying the remainder by the "constant" of damping, you get the loss of hearing in sensation units. By using a number of forks of different pitches, one obtains a curve similar to that derived from the audiometer, but it is true that doing this with forks requires more time.

We thought at first that owing to their light mass the magnesium alloy forks would not be adaptable for bone conduction. Action and reaction are equal, but opposite in direction, and when a heavy fork comes in contact with the skull it, so to speak, pushes the skull away, and the latter then presses against the fork with equal force. Much to our agreeable surprise, it was found that these magnesium forks served very well for testing bone conduction, even though the bone conduction is not as long as that for steel forks, especially when they are placed on the vertex. Forks as usually employed, range in pitch from C-2 (16 double vibrations) to c-6 (8,192 double vibrations). A 12 double vibration fork is made, but it is very large and difficult to manipulate. Forks of higher pitch are also constructed but not for general otologic practice.

With reference to overtones, it should be remembered that when the forks are struck not only the fundamental is sounded but a number of overtones, the first of which is very loud

and has a vibration period usually five or six times that of the fundamental, and this often causes very disturbing results. To eliminate overtones, weights are attached to the prongs of the fork, but these of course damp it so that it vibrates a much shorter period of time. There are some very fine unweighted forks, such as the Bezold A (108 double vibrations) and a-1 (435 double vibrations), made by Edelmann, whose overtones persist but a few seconds after striking them. Bezold himself has called attention to the fact that if weights are not provided, the overtones may be eliminated by placing about each prong a wide rubber band. This does not add materially to the weight of the fork and does not shorten the period of vibration to the extent that the heavy metal weights do. When the unweighted magnesium forks are struck, they usually give forth rather shrill and persisting overtones, and this has been commented on by a number of men to whom these forks have been demonstrated. However, if the weights be removed from the medium pitched Edelmann forks it will be found that they also have very disagreeable overtones. By merely slipping upon the prongs the above mentioned piece of rubber tubing, these overtones are easily obviated. With the Edelmann series of forks, by the use of weights and with markings upon the prongs, it is possible to set the weights at various points and thus get six or seven pitches from one fork. The three lowest pitched magnesium forks are also supplied with weights, and the prongs are calibrated so that it is possible to obtain a range of a whole octave from one fork. This is not only a very convenient but very economical provision, as it saves at least one or two forks in the series when an attempt is made to get a continuous range for the lower octaves.

In order to get comparative findings of magnesium alloy forks as compared with the Edelmann series, careful examinations of a considerable number of patients were made. I am greatly indebted to my associate, Dr. Norman Leshin, who carried out accurate tests on normal and pathologic cases.

The summary of these tests is as follows:

I. BY AIR CONDUCTION.

C-1 (32 double vibrations) magnesium fork is heard slightly longer than the steel, its average being about 25 seconds.

C (64 double vibrations), magnesium fork was heard almost twice as long as the steel fork (both being weighted), the average being 45 seconds.

c (128 double vibrations), magnesium was heard about the same length of time as the steel, an average of 75 seconds.

c-1 (256 double vibrations), steel fork was heard about ten seconds longer than the magnesium, the average being 75 seconds.

c-2 (512 double vibrations), steel had a duration of from 20 to 25 seconds longer than the magnesium, which averages 55 seconds.

c-3 (1,024 double vibrations), steel forks are heard almost twice as long as the magnesium, but since the latter has a duration of 45 seconds, it vibrates long enough.

c-4 (2,048 double vibrations), steel forks are heard twice as long as magnesium forks which have an average of about 20 to 25 seconds.

c-5 (4,096 double vibrations), steel fork was heard from two to three times as long as the magnesium fork, which has a duration of from 7 to 10 seconds.

We are hoping that Mr. Eisenhour will succeed in modifying the c-5 (4,096 double vibrations) magnesium fork in order that the period of vibration may be lengthened so that it will then more favorably compare with the Edelmann fork of the same pitch.

In a word, we may say that so far as hearing by air conduction is concerned, the magnesium forks serve just as well as steel ones, with the exception of the highest tones, namely, c-4 (2,048 d. v.) and c-5 (4,096 double vibrations). However, in those cases where it is desirable simply to find out whether the individual can hear a tone of that pitch, magnesium forks will serve very well, but where we wish to test the actual duration of hearing by air of these high tones the steel forks give better results.

### II. BY BONE CONDUCTION.

Some of the medium pitched magnesium forks do not give quite as satisfactory results as do the steel forks of the same pitch, when set upon the vertex, but where they are placed

upon the mastoid region, as in the Rinne test, the magnesium forks of such pitch as c-1 (256 double vibrations), and a-1 (435 double vibrations), are just as useful as the steel forks of the same pitch. For instance, the c-1 (256 double vibrations) magnesium fork serves just as well as the a-1 (435 double vibrations) steel fork in doing the Rinne test, because with it there is a difference between the hearing for air and bone conduction of thirty seconds or more. The Weber test may easily be carried out with one of the lower pitched magnesium forks such as C (64 double vibrations). When the Schwabach test is performed by placing the fork on the vertex, a steel fork, such as the unweighted A (108 double vibrations), is probably the best type. As I have pointed out in previous papers, many otologists determine duration of bone conduction by way of the mastoid, thus using the bone conduction factor of the Rinne test for that purpose. When this is done, magnesium forks, as previously mentioned, serve just as well as steel forks of the same pitch. When forks of similar pitch are used, bone conduction is usually about 50 per cent longer by way of the mastoid process than by way of the vertex, to which fact I called attention quite a number of years ago in "The Study of the Schwabach Test."

Whether the magnesium forks will ultimately to a considerable degree displace steel forks in otologic practice cannot be positively stated at present; for it will require considerable experience on the part of many otologists to settle this question definitely, especially with reference to the highest tone, c-5 (4096 double vibrations). It seems, however, that we have here quite a satisfactory solution of the problem of supplying forks made of a metal which is rustproof, and whose weight is such that long continued testing does not prove as fatiguing as it does with the heavy steel forks. Hence these new forks have some very decided and desirable advantages to their credit.

## CONCLUSIONS.

1. An alloy consisting of 95.6 per cent magnesium, 0.4 per cent manganese and 4 per cent aluminum serves very well for the manufacture of a complete tuning fork series, from C-2 (16 double vibrations) to c-5 (4,096 double vibrations).

2. Magnesium alloy forks seem in actual practice to be no more brittle or liable to breakage than the steel forks, when properly handled.

3. These new forks are very light in weight so that they can be used for long periods of time without tiring the fingers.

4. The price of these forks manufactured in this country is about 25 per cent less than that of the best imported German forks.

5. Magnesium compound forks are rustproof, and therefore do not change their pitch on exposure to moisture or to manipulation with the hands.

6. The highest pitched c-5 (4,096 double vibrations) magnesium fork does not vibrate long enough for all purposes, but the other forks of the series in actual practice serve very well both in testing air and bone conduction.

7. When provided with the "constant" of damping, these forks as well as steel forks can be used to obtain the same curve and the loss of hearing, figured in sensation units, as is done with the audiometer.

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LXIX.

# NONTUBERCULOUS LESIONS OF THE LARYNX IN THE TUBERCULOUS.*

By Joseph B. Greene, M. D.,

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In considering nontuberculous lesions of the larynx in the tuberculous we must bear in mind the very great frequency of laryngeal tuberculosis as a complication of pulmonary tuberculosis. It is generally accepted that this complication occurs in at least 25 per cent of all cases, and in the more advanced stages of the disease the percentage is somewhat higher. According to Fetterolf, in a series of 100 autopsies of patients dying of pulmonary tuberculosis, 83 per cent showed positive microscopic evidence of laryngeal tuberculosis. Bearing in mind the frequency of laryngeal tuberculosis in the tuberculous, we should think first of this complication when symptoms are directed to the larynx, particularly so if there is pain or hoarseness. In other words, the case should be regarded as tuberculous until it is proven otherwise.

However, a patient suffering from pulmonary tuberculosis possesses no immunity from other laryngeal affections, but if one takes a careful history of the case, noting the situation and appearance of the lesion, together with the age and sex, it is seldom that one will err in reaching a correct diagnosis. In this discussion I shall leave out of consideration such rare affections of the larynx as actinomycosis, chondroma, leprosy and lupus, none of which I have encountered, but shall speak only of those conditions generally met with in special practice.

The most frequent nontuberculous lesion met with in the tuberculous is undoubtedly simple catarrhal laryngitis. According to Lockard, the tuberculous patient is especially subject to this type of laryngeal affection, probably due to irritating cough, bad environment and the unhygienic living which

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precipitated the pulmonary attack. It has been my observation that the "rest cure" places a heavy burden, so to speak, on the nasal mucous membranes by its effect on the local circulation, inducing an obstructive condition of the nose. This obstruction can readily be a predisposing cause of simple laryngitis. In cases presenting symptoms of acute simple laryngitis it is at times impossible to make at once a positive diagnosis, but the patient should be placed on silence and simple therapeutic measures instituted, until sufficient time has elapsed, usually a week or so, when a positive diagnosis can ordinarily be made. It is not a waste of valuable time, as the treatment of the two conditions at this stage is practically the same. However, if the redness in the larynx is localized in certain portions of the larynx, as in one cord, or if there be noted certain areas of red infiltration, we should strongly suspect a tuberculous complication. We should bear in mind the strong predilection of laryngeal tuberculosis for the posterior part of the larynx—the commissure and ends of the cords. Chronic catarrhal laryngitis in the tuberculous may offer some diagnostic difficulties. However, a close inspection of the larynx, if it be tuberculous, will usually show an asymmetrical character of the lesion, whereas, the nonspecific type of disease shows a uniform involvement of the interior of the larynx. The symptom of hoarseness is less marked and pain is rarely present in the simple type of affection. Should there be present a true erosion, which can be easily overlooked, especially when located on the posterior surface of the epiglottis, we must regard the affection as other than a simple catarrhal laryngitis.

Pachydermia laryngis is a type of chronic laryngitis which from its posterior location may at times simulate tuberculosis of the larynx. It differs from tuberculosis, however, in that it tends to show a prominent excrescence on one cord with a corresponding depression at times on the opposite side; a state of affairs not met with in tuberculous laryngitis. There is also usually present some associated infiltration of the posterior commissure which bears a close resemblance to a tuberculous lesion, but the surface presents a more even appearance and there are no signs of true ulceration. A history of cases of pachydermia usually reveals some abuse of the voice, and

there may have been some chronic infection of the sinuses and tonsils. The voice is usually only slightly affected, if at all, in contrast to the tuberculous lesions, and pain is not present.

Cysts are interesting and rare conditions of the larynx and are usually discovered by accident. They only produce symptoms when large enough to cause obstruction or when so situated as to interfere with phonation. The usual situation for such growths is on the anterior surface of the epiglottis. The three cases which I have observed were all in this locality and were in tuberculous individuals. The appearance of these cystic growths is so characteristic that no difficulty in diagnosis is encountered.

Recurrent nerve paralysis causing immobility of one cord, usually the left, occurs in the tuberculous and may be due to pressure from pleuritic adhesion. However, Wood, in reviewing the work of Manassa, attributes this lesion of the recurrent nerve to pressure of trachea-bronchial glands. Against this view is the frequency of glandular involvement in children and the infrequency of recurrent nerve paralysis in the very young. The chief symptom produced in these cases is hoarseness, which is easily explained on laryngeal examination, provided no tuberculosis of the larynx is present. Four cases of this type have occurred in my practice, three being in women, on the left side of the larynx, and one in a man, involving the right recurrent laryngeal nerve. We must bear in mind, also, the occurrence of paresis of the recurrent nerve due to aneurysm, enlargement of the heart, particularly the right auricle in mitral stenosis, and mediastinal tumors.

Papillomata, the most frequent nonmalignant laryngeal growth, usually has its inception in childhood, when laryngeal tuberculosis is almost unknown. In any event, the appearance of these noninflammatory lesions, usually of the cords, is so characteristic that the diagnosis offers little difficulty. Hoarseness is the chief symptom and obstruction takes place only where the growths involve an extensive area of the larynx. Vocal nodes, situated at the juncture of the anterior third of the cords with the posterior two-thirds, have only hoarseness as a symptom. This may suggest a tuberculous complication,

but the appearance usually of two small corresponding nodules on the anterior part of the cords makes the diagnosis comparatively easy. In one of my two cases a careful history of the case, so important in all laryngeal conditions, excluded the possibility of the hoarseness being due to a tuberculous larynx. The patient was a woman of thirty-five, a sufferer from pulmonary tuberculosis for a period of five years, who had complained of hoarseness since the age of thirteen. Examination of the larynx confirmed what the history strongly suggested, that the lesion in the larynx was not tuberculous in character. The other case was the association of vocal nodes in a young woman of twenty-five who had also a characteristic tubercular lesion of the posterior part of her larynx.

Syphilis of the larynx occurring within the larynx proper has so many points of resemblance to tuberculosis that we are at times confronted with difficulties in reaching a positive diagnosis. In this connection Sir St. Clair Thomson says there is no difficulty in distinguishing tuberculosis from secondary syphilis or a simple gumma, but with ulcerating syphilis the task is not so easy. Fortunately, however, syphilis of the larynx is of infrequent occurrence. As Spencer wisely says, the history in such cases is of almost no value. However, the Wassermann test comes to our aid, though we must bear in mind its limitations.

Barring such unusual conditions as leprosy, yaws and acute malarial fever, a positive Wassermann on repeated examinations means syphilis. However, this is not proof positive that the lesion in the larynx is syphilitic. A negative Wassermann means little, for the test may be vitiated by the previous ingestion of alcohol or the administration of antisyphilitic treatment.

Syphilitic ulcers occurring in the larynx are deep and crater-like with a foul necrotic base, as distinguished from the tubercular lesion, which is superficial in character with an irregular border having a base covered with granulations. Syphilitic lesions occur on the anterior surface of the epiglottis, while tuberculosis usually attacks the lingual surface or the upper margin. Deep ulcerative lesions in the upper larynx, particularly if there is present a red zone surrounding this necrotic

area, suggests syphilis rather than tuberculosis. Syphilitic lesions of the arytenoid are characterized by a red inflammatory swelling which soon shows deep, craterlike ulceration. This is unlike tuberculosis of this region, which usually attacks both arytenoids, in which case ulceration, if present, occurs late and is superficial in character. There has seemed to me a striking tendency for syphilis to attack the tissues at the root of the tongue, which is not so in tuberculosis.

In reference to pain, it has been my impression that this symptom has very little diagnostic value. If the ulcerative lesion occurs on the epiglottis or the arytenoids, the pain may be quite as severe in syphilis as in tuberculosis. Lesions within the larynx proper are not particularly painful, as a rule, whether they be tuberculous, syphilitic or cancerous. Recently a man, aged thirty-six, with moderately advanced pulmonary tuberculosis, came to me complaining of severe pain on swallowing which interfered with his taking food. This had lasted for several weeks. Examination showed slight swelling of the epiglottis with general redness of the interior of the larynx. There was noted also an ulcer on the left side of the root of the tongue. The appearance of the lesion suggested syphilis, which was confirmed by the finding of a 4 plus Wassermann and relief of symptoms following a few doses of antisyphilitic treatment. Finally, it may be necessary to resort to the use of the therapeutic test of antisyphilitic treatment in making a diagnosis of syphilis from tuberculosis. It has been considered by some unsafe to administer arsenic preparations to patients suffering with pulmonary tuberculosis, though this has not been our experience when caution is used in dosage to avoid too violent systemic reaction.

The cases which give us most concern are those of suspected cancer in the tuberculous, but fortunately such puzzling cases are rare, for laryngeal tuberculosis is a frequent complication, while cancer is rarely met with in the tuberculous. It is only in early or moderately advanced cases of pulmonary tuberculosis that a positive diagnosis of cancer is important, for obviously the advanced cases would not be suitable for surgical removal. In making a diagnosis we must bear in mind that cancer is most frequently found in those past forty years of age, while tuberculosis occurs most frequently in those

under forty years of age. In a series of 385 cases of cancer occurring at the Mayo Clinic, reported by New, 95 per cent were more than forty years of age. One case, however, occurred in a boy of fifteen. Cancer of the larynx is rare in women, which is not the case in tuberculosis.

Intrinsic cancers, and benign tumors as well, are most frequently found on the anterior part of the cords, while tuberculosis shows a decided preference for the posterior ends of the cords, the commissure and arytenoids. In the great majority of cases of intrinsic cancer the first symptom is hoarseness, whereas in tuberculosis the patient's first complaint may be slight discomfort referred to the larynx and increase of cough. When the tuberculous lesion is confined to the cords pain is not a feature, either in tuberculosis or cancer. Aside from the location previously referred to, there is often a distinct difference in the appearance of the two lesions; but cases do arise which tax one's skill to the utmost in making a correct diagnosis. Even in the early stages of laryngeal tuberculosis there is noted some inflammatory redness, in contrast to incipient cancer. There is present at times a general redness of the larynx. According to Krause this inflammatory reaction is due to sensitization (allergy) to the products of the tubercle bacillus.

Tuberculomata is the type of lesion which most resembles cancer, but fortunately this form is not often encountered, and when found is usually on the posterior half of the larynx. Cancer in its earliest stage is usually a noninflammatory, warty growth on the cord, producing no symptoms except that of huskiness or hoarseness. It later develops into an irregular cauliflower mass, with all the characteristics of malignancy. At this stage it could hardly be mistaken for tuberculosis. Impaired mobility of the cord is not observed in cancer until the deeper structures are involved, so this symptom, so often referred to, should not be looked for in the early stages of cancer. However, in the deeper type of laryngeal cancer, which MacKenty describes as "subsurface," defective movement of the cord is an early symptom. In tuberculosis, impaired mobility of the cord is not an early symptom, though it does occur late in the disease, when the diagnosis is relatively easy.

Extrinsic cancer involving the epiglottis, arytenoids or aryteno-epiglottic folds is relatively rare, and has few points of resemblance to tuberculosis. In case the arytenoid is involved, it is usually confined to one side, whereas tuberculosis attacks both arytenoids. Pain is not considered a symptom of cancer in this region, but when ulceration takes place it has seemed to me to be quite as severe as in tuberculosis. Cancer may be grafted on a previous tubercular lesion, which changes the entire picture.

The question of biopsy in suspected cancer of the larynx has been under discussion for a number of years, but no definite conclusion has been reached, except that it is not wise to delay operation too long, if the microscope shows positive evidence of cancer. It is my conviction that a biopsy is rarely necessary or wise in reaching a diagnosis between cancer and tuberculosis in the frankly tuberculous individual. It is an accepted fact that the results of microscopic findings are not infallible, in that a negative finding as to cancer means very little, and also that a positive finding may occasionally be made in a nonmalignant growth. In the final analysis, as MacKenty says, we must rely upon the appearance and clinical course of the disease made after days of careful study and observation. In the intrinsic type of lesion, undue haste in making a diagnosis is not required.

In my experience over a number of years, it has been necessary in only one case of pulmonary tuberculosis to have a biopsy to determine the presence or absence of laryngeal cancer. This patient, a man of sixty-two, had suffered from pulmonary tuberculosis for a number of years, at times complicated with hemorrhage. His blood Wassermann was negative. He came to me in May, 1927, with a diagnosis of laryngeal tuberculosis. When seen by me somewhat later, there was noted a large red swollen arytenoid on the left side, which extended forward, involving the left vocal band, obscuring the left cord. There was immobility of the left side of the larynx. The right side of the larynx was normal, no swelling of the right arytenoid being present. The epiglottis was normal. There was present severe pain on the left side of the larynx, radiating to the ear. The arytenoid presented on sev-

eral occasions peculiar elevated pustules containing a drop of exudate, which required puncture with the cautery needle.

This unusual condition of superficial pustules I have never seen associated with tuberculous arytenoids. This was evidently a new picture on the screen, so to speak, and was not characteristic of tuberculosis or any lesion of the larynx with which I was familiar. Dr. MacKenty saw the case in consultation and thought it probably cancer and advised a biopsy. This was done, but neither cancer nor tubercle was made out. A second specimen was removed four months later, probably from a different region of the tumor, which revealed positive evidence of cancer. The larynx was removed a few days later by Dr. MacKenty, and microscopic section revealed both cancer and tubercle. This case was interesting in that there was a double infection of cancer and tubercle, and the fact that the first biopsy was entirely negative, showing the importance of clinical findings as well as a microscopic diagnosis.

Four cases in frankly tuberculous patients have come under my observation in recent years, on whom biopsy had been done for diagnosis, having been under suspicion of malignancy. The ages of these four patients, they being respectively 28, 33, 35 and 45, should have lent weight on the side of tuberculosis as against cancer. Microscopic examination of the specimens removed in each of these four cases only established the diagnosis of tuberculosis, which could have been made by other means. It has seemed to me that a careful study of the larynx of these cases over a period of time, as stressed by MacKenty and Thomson, together with a careful chest examination by a competent internist, with stereoscopic X-ray plates, would have spared these patients the discomfort and probable harm incident to the biopsy. Inasmuch as three of the cases were originally intrinsic, and all later became extrinsic, developing active tuberculosis of the epiglottis, there is suggested the possibility that trauma of the laryngeal speculum played a part in the spread of the infection. When the necessity arises that a specimen must be removed from the larynx of a tuberculous individual for diagnostic purposes, it appears preferable to use the indirect method, followed immediately by searing the raw surface with the electrocautery.

## LXX.

# EDUCATION OF THE GENERAL PHYSICIAN AND PUBLIC TO THE SINUS PROBLEM.

By H. B. Lemere, M. D.,*

Omaha.

The recognition by the laity of the existence of nasal sinus disease is evidenced by the number of persons who are aware that their symptoms are due to sinus disease. Equally common is the popular prejudice against any form of sinus surgery. People are aware that their nasal or postnasal catarrh means sinus trouble, but they have heard for a long time that no medical treatment, either local or general, will bring them much relief and that operative interference is a calamity calling for repeated operations resulting in no relief of their symptoms and occasionally in death. When told a sinus or nasal operation is advisable the patient almost invariably states that he has been advised against this course by his friends and not infrequently even by his physician.

This pessimistic attitude is undoubtedly hampering our effectiveness in dealing with upper respiratory infections. It is partly due to popular ignorance as to what the term sinus trouble means in terms of anatomy and physiology. Popular articles and talks with our patients will supply the elementary knowledge necessary to correct this ignorance. The present popular mental attitude toward sinusitis is a case of too little knowledge being a dangerous thing.

It is true that with that form of suppurative sinusitis which demands repeated operations and very radical measures the patient's pessimistic attitude is almost warranted, and it is generally these cases which are used as examples by patients and even general physicians in forming their opinions.

Fortunately, the great majority of infections of the upper respiratory tract are nonsuppurative and lend themselves well

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to treatment or surgery. I may be pardoned perhaps if I quote from a paper I presented to this section in Colorado Springs, in 1920, as the opinion which I then expressed I have found no reason to change:[1]

"I have been surprised to find constant evidence of antrum infection. Almost without exception those cases which I have supposed to be ethmoid disease showed the main infection to be antrum disease. The appearance of the middle fossa with the swollen middle turbinate, mucus and polyps, which is usually diagnosed as ethmoiditis, I have so frequently found associated with antrum involvement that I think most of the cases of ethmoiditis are dependent on an underlying antrum involvement."

Dr. Fred Stauffer states, in a recent article:[2] "I do not wish to convey the idea that all chronic suppurative conditions of all the nasal accessory sinuses can be cured by drainage of the antrum—I do want to emphasize that this method is applicable to and will cure a great many cases of chronic suppurative ethmoiditis with polypous formation that cannot be cured without drainage of the maxillary sinus."

This opinion seems from the literature to be more and more prevalent.

Perhaps part of the pessimism of our patients is caused by those who, seeking relief from symptoms which could only come from involvement of a large sinus, are subjected to submucous operations for inconsequential deflections of the septum or exenteration of ethmoid cells, often with no relief of their symptoms. They have undergone an operation, minor from the viewpoint of the surgeon but of major magnitude to the patient's mind. If they do not receive relief they constitute another recruit to the army of malcontents with nasal surgery. I say this with all humility, as I myself have been disappointed in the results of such procedures in my own operative work, at times perhaps, getting a more classical interior to the nose but slight, if any, improvement from the patient's standpoint.

After treatment has shown a nonsuppurative condition of the antrum to be chronic and persistent, should we refuse to clear up the condition by operation because we cannot demonstrate free pus? The general surgeon is amply justified when

he removes an appendix which, while showing signs and symptoms of appendicitis, yet has no abscess or free pus present. His diagnosis has been based on the symptoms of a low grade infection, and it is considered sound surgery. In several nonsuppurative maxillary sinus cases I employed Baum's method of diagnosis, using, however, a smaller trocar and Wappler's antroscope. Before using the antroscope I obtained through the canula a swab which I placed in brain broth culture and found these sinuses invariably infected, generally obtaining some form of streptococcus. When the clinical symptoms are plainly evident, I do not now consider it necessary to obtain such verifying information as this procedure, however edifying to the surgeon, is not without discomfort to the patient. For those who are uncertain of the existence of infection in nonsuppurative nasal conditions the use of Baum's method will be both instructive and convincing.

In order to find how far I was relieving the symptoms of my patients in whom I had diagnosed nonsuppurative antrum infection, I wrote a circular letter containing questions to several on whom I had operated. I received twenty-seven replies. The letter, with the replies tabulated, is as follows:

I am interested in knowing the result of the operation I performed on you and would greatly appreciate your answer to the following questions:

1. Do you catch cold as easily as before the operation, and if so, does it last as long:
   No, 20; yes, 6; more, 1.
2. Have you noticed more energy since your operation?
   Yes, 22; no, 5.
3. What other symptoms troubled you before the operation?
   Discharge from nose, headache, etc.
4. Have you been relieved, and is your general health better?
   Yes, 24; no, 3.

The unfavorable replies were scattered so that 99 per cent expressed themselves as being benefited in some way. The one who replied unfavorably was a case of obscure diagnosis. On examination by Baum's method, several white tubercles could be seen in the antrum, and though we were never able to demonstrate a tuberculous infection while he was under my care there was some underlying general condition I felt sure

was the cause of his afternoon temperature, headaches and underweight.

I did not send this letter out for the purpose of publication or statistics. I merely wished to check myself on my results, and am inserting it in this paper on account of the bearing it has on the popular statement that a patient is worse after a sinus operation than he was before and that repeated operations were always necessary.

The prejudice against operative work will, other things being considered, be in direct proportion to the failures. The failure of an intranasal window resection comes mainly from a closure of the window. Often the probe indicates that a large window has been secured, when on careful inspection it is seen that one has only produced a break with a large swinging flap which on healing will completely close the opening. It is necessary to be sure that the wall is completely bitten away. Most of the rasps break the wall without removing it, and give the impression that there is a large aperture when in reality merely a flap has been made. Many times the inferior turbinate swells and heals over and occludes the opening. It is better to sacrifice enough of the turbinate to insure a permanent and accessible opening.

I have had not a few patients of my own and other operators complain of a return of their symptoms in whom I have found an adequate antral opening but in whom adequate nasal hygiene had not been carried out. These cases cleared up easily under irrigation and proper nasal hygiene without further operative interference. Lillie believes that a so-called waterlogged condition of the mucous membranes results from the use of washes. The mucous membrane of the nose is so often swollen and the seat of polypoid or mucoid swelling in instances where no operation has been done and where no washes have been used that it is necessary to look for further explanation of this condition. As mucoid swelling over the turbinates and polyp formation usually subside on permanent aeration and drainage of the antrum it is possible that the cause for this condition lies in the infective and irritative discharge from the antrum. My personal experience does not extend to any case of mucoid or polypoid swelling of the mucous membrane which was due to washing. I have seen such

cases where the washing was inadequately carried out and heavy secretions remained in the antra. On properly washing out these cavities the swelling subsided. I wonder if Lillie's success in those cases in which he stopped the irrigation did not result from his skillful treatment in removing the secretions and perhaps from the possibility that the irrigations had been ineffectual in removing the thick irritating secretions. Many patients cannot always be within reach of a skilled rhinologist, and it is necessary to instruct them how to clean out their own sinuses during a temporary reinfection. The only method I know by which a patient can remove this occasional thick secretion is by irrigation, and under careful instruction this seems to produce very happy results.

Much dissatisfaction, I am sure, comes from the expectation of immediate relief after an operation and the lack of appreciation of the essential chronicity of these cases. After aeration and drainage of the cavities it is somewhat rare to find a prompt return to normal. The improvement increases after weeks, months or even years, and it is better to so inform the patient before operation, or the credit which belongs to the operation will be given to something inconsequential which has happened in the meantime.

In conclusion I think it advisable to meet the situation by education of the general profession and of the people; that in the majority of cases, nonsuppurative in character, a permanent and accessible opening under the inferior turbinate will assure the patient that his fears of repeated operation will not be realized; that instruction in nasal hygiene is necessary to obtain the best results; and that explanation should be made that the full benefit of the operation may be delayed for months.

## REFERENCES.

1. Lemere, H. B.: Transactions of the American Laryo-Rhino and Otol. Soc., 1920.

2. Stauffer, Fred: Annals of Otology, Sept., 1928, p. 845, No. 3. Vol. 35.

LXXI.

# THE FUNCTION OF THE MUSCULAR ATTACHMENTS OF THE TONSIL.

By Homer A. Trotter, M. D.,

Buffalo.

The effect upon the tonsil, with emphasis on the part that the muscular attachments play in their function, is apparently little understood. However, careful observation of the muscular behavior and its effect upon the tonsil is convincing that the adjacent musculature has a positive function.

Observation and study have influenced the belief that the tonsil has an intrinsic and extrinsic function assisted by its muscular attachments. That the physiology of the tonsil can be understood, a brief description of the anatomy is necessary.

The tonsil is essentially lymphoid tissue and is a member of the lymphoid ring which includes the thymus, spleen, lymph glands, pharyngeal and lingual tonsils and the appendix. The location of the tonsil in the oral cavity exposes the organ to infection, and the arrangement of the organ, its pouches and crypts, is especially adapted to the entrance of infection.

The exposed surface of the tonsil consists of stratified, squamous epithelium which lines irregular tubes or crypts which dip down into the organ and are numerous. The crypts and pouches play an imporant part in the physiology of the tonsil in that the area of mucous membrane is increased to assist phagocytosis. The tonsil is separated from its bed by the pharyngeal fascia, which sends in trabeculæ and consists of the main framework of the organ. The bulk of the tonsil is made up of lymphoid tissue of the diffuse variety and contains solitary follicles which show germinal centers and are found chiefly around the crypts. Lymphocytes can be observed *enroute* toward the crypts, where they function as phagocytes. Phagocytosis takes place in the lymphoid tissue and in the crypts of the tonsil.

The products of phagocytic action are eliminated by the lymphatic vessels or extruded into the crypts as exudate and later removed by the musculature adjacent to the tonsil by means of pressure. In other words, the extrinsic function is the process by which contraction exerts pressure upon the tonsil. The force disgorges or expells the contents of the crypts or pouches into the oral cavity and is really an opening and drainage of the organ.

The plica semilunaris in the fully developed tonsil represents the tonsillar hood. The edge of the orifice is guarded, except near the posterior pillar, by folds of the plica, arching from the front to the back. The physiologic significance of the plica semilunaris is that it exerts a traction from the upper pole of the tonsil during the tonsillar excursion caused by the muscles in the state of contraction, thus producing an eversion of the organ which assists to disgorge or expel the crypt contents.

The tonsillopharyngeus muscle is attached to the groove at the junction of the middle and lower third of the tonsil and inserted by numerous connective tissue fibers to the sheath of the palatopharyngeous. The physiologic significance of this muscle is that it holds the tonsil in position and supports the nerves and blood and lymphatic vessels. Its action pulls the lower pole of the tonsil inward.

The palatoglossus muscle is an independent bundle of muscle fibers that bounds the tonsillar bed in front. It has a fan-shaped origin in the oral surface of the soft palate and is arranged around the throat, forming the prominence of the anterior pillar of the fauces and terminating in the lateral aspect of the tongue. It (1) raises the back of the tongue, (2) narrows the fauces, (3) assists to compress the tonsil laterally, (4) depresses the soft palate, and is the (5) anterior support for the tonsil.

The palatopharyngeus muscle is the most important muscle in relation to the tonsil. It is an inner sheet of muscle fibers disposed vertically and forms a continuous layer around the pharynx between the submucosa and the superior constrictor. Above its fibers are attached to the soft palate, the eustachian tube and the base of the skull. Below, its fibers lose them-

selves in the upper esophageal wall. It has a slight reinforcement from the stylopharyngeus, though most of the stylopharyngeus fibers remain separate. The lateral part of this muscle interests us most. This lateral part arises from the soft palate as far laterally as the hamular process. Its fibers, originally a thin sheet above, become thinner in their descent, reinforced by and intermingled with, some penetrating fibers of the stylopharyngeus, thus forming the posterior pillar. It (1) draws the pharynx upward, (2) depresses the soft palate, (3) narrows the fauces, (4) assists to compress the tonsil laterally aided by the anterior pillar, (5) assisted by the superior constrictor forces the tonsil outward toward the median line of the fauces.

The superior constrictor is a muscle with transversely disposed fibers. It forms the circular musculature of the pharynx and lies between the inner vertical fibers of the palatopharyngeus and the outer vertical fibers of the stylopharyngeus. It arises from the lower third of the internal pterygoid plate, the hamular process, the pterygomandibular raphe and the mandible at the attachment of the raphe, which lies between the superior constrictor and the buccinator. Passing transversely, the superior constrictor terminates in the median raphe on the back of the pharynx, its upper fibers inclining upward to the base of the skull. The muscle has free, sickle-shaped upper and lower margins. Above the muscle the levator palati reaches the pharynx, and between the lower border of the superior constrictor and upper border of the middle constrictor the fibers of the stylopharyngeus penetrate. Its action is (1) compression of the pharynx; (2) in conjunction with the palatopharyngeus, it assists to compress the tonsil, forcing the tonsil outward toward the median line of the fauces.

Peripheral stimulation of the nerve end organs located in the tonsil find their way to the medulla by two routes, the posterior palatine nerve, through the autonomic ganglion, known as the sphenopalatine, and then by way of the sensory fibers of the trigeminal nerve to the nucleus of the fifth nerve in the medulla. The other nerve pathway is by means of the afferent neuron of the glossopharyngeal, through the jugular and petrosal ganglia to the nucleus tractus salitarius in the medulla.

By means of associated neurons the nucleus of the fifth nerve is brought in direct relationship with the nucleus ambiguus, which is the motor center of the vagus and spinal accessory nerves. The nucleus tractus solitarius is also brought in contact with the nucleus ambiguus through associated neurons.

Impulses generated in the nucleus ambiguus and bulbar part of the nucleus of the spinal accessory nerve are sent to the pharyngeal plexus located in the posterior pharyngeal wall by means of the vagus and spinal accessory nerves. From the pharyngeal plexus efferent neurons pass to the responding muscles, which are the tonsillopharyngeus, palatoglossus, palatopharyngeus and superior constrictor.

The neural anatomy involved is obviously a reflex arc which makes possible the act of deglutition and gagging. Deglutition, therefore, exerts compression upon the tonsils by means of the adjacent muscles and thus assists in the extrinsic physiologic function of the tonsil.

During the act of deglutition, gagging or retching, it is possible to measure the muscular effort exerted upon the tonsils by means of a specially built rubber balloon which is placed between the tonsils and, when compressed, registers on a sphygmomanometer the amount of muscular effort in millimeters.

It has been found in a number of measurements that the muscular pressure is 56 to 60 mm. in deglutition, 80 to 86 mm. in gagging and 90 to 100 mm. in retching.

FAUCIAL MEASUREMENTS IN MM.

| | At Rest | Contraction | Excursion |
|---|---|---|---|
| R. B. | 28 | 10 | 18 |
| R. D. | 26 | 14 | 12 |
| L. H. | 26 | 10 | 16 |
| F. B. | 25 | 12 | 13 |
| J. S. | 30 | 11 | 19 |

It is possible to measure the muscular excursion by means of reversed calipers, and the following measurements are the average of a hundred fauces: Faucial diameter at rest, 26 mm.; muscular contraction, 14 mm. Therefore, the average muscular excursion has been found to be 12 mm.

MUSCULAR EFFORT, MEASURED IN MM. OF MERCURY.

| | Swallowing | Gagging | Retching |
|---|---|---|---|
| R. B. | 60 | 86 | 100 |
| R. D. | 56 | 82 | 92 |
| L. H. | 58 | 84 | 96 |
| F. B. | 56 | 85 | 94 |
| J. S. | 57 | 86 | 98 |

It has been possible to secure a tracing showing a curve caused by gagging, indicating the muscular effort upon the tonsil, graphically, also the time in seconds. Obviously, the amount of muscular work exerted on the tonsil by the combined musculature is sufficient to compress the pouches and crypts, causing an emptying of the contents, which may be performed at each deglutition.

## CONCLUSION.

It has been observed by stimulation of the nerve endings or terminals in the mucous membrane of the fauces, the pharynx and the tonsil, that a reflex action occurs which affects the muscles adjacent to the tonsil.

By means of the conjoined contraction of the muscles, associated with the tonsil which cross and intersect intricately, pressure is exercised upon the upper pole of the tonsil, causing this portion to move outward and slightly forward toward the median line of the fauces. Pressure is also exerted upon the tonsil laterally by means of the palatoglossus and palatopharyngeus muscles.

The contraction of the tonsillopharyngeus muscle, which occurs simultaneously with the upper musculature, directs the movement of the tonsil inward and this causes the tonsil to tip.

The traction caused by the plica semilunaris in the tonsillar excurison and deviation from the vertical plane affects the upper portion of the tonsil, which causes a mechanical eversion of the organ, producing a mechanical expelling action or disgorgement of the tonsillar pouches and crypts.

By this method the tonsil throws off the excessive exudate, food particles, cellular débris and bacteria and frees the pouches and crypts of foreign material and promotes drainage.

40 W. North St.

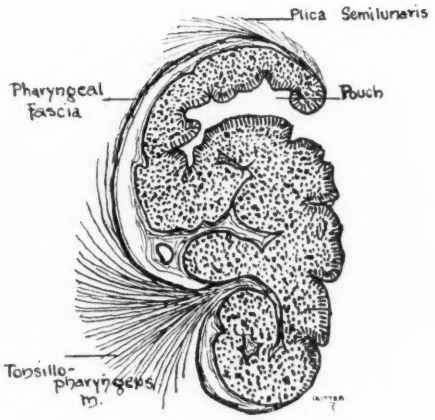


Fig. 1. Tonsil at rest.

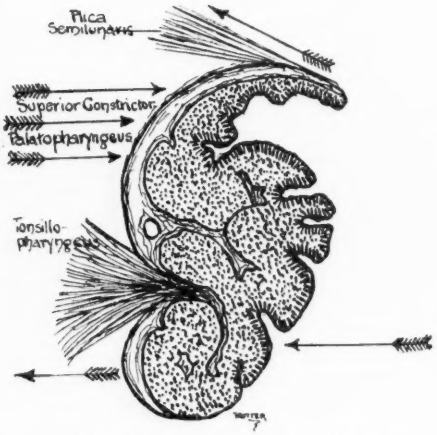


Fig. 2. Tonsil—Muscular contraction.

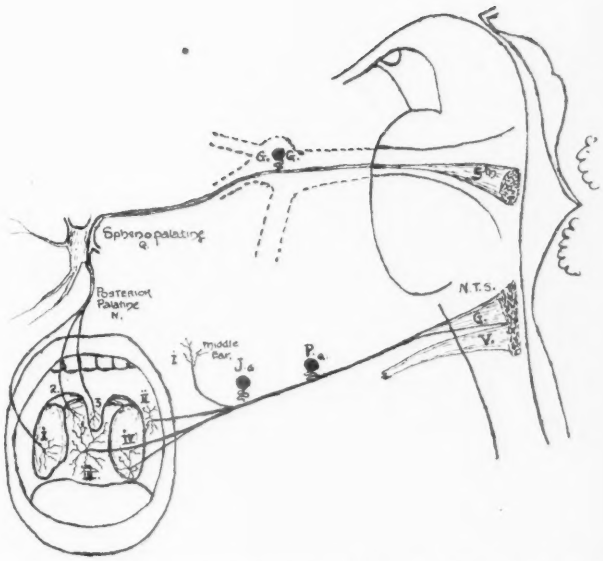


Fig. 3. Primary terminal nuclei of afferent glossopharyngeal.

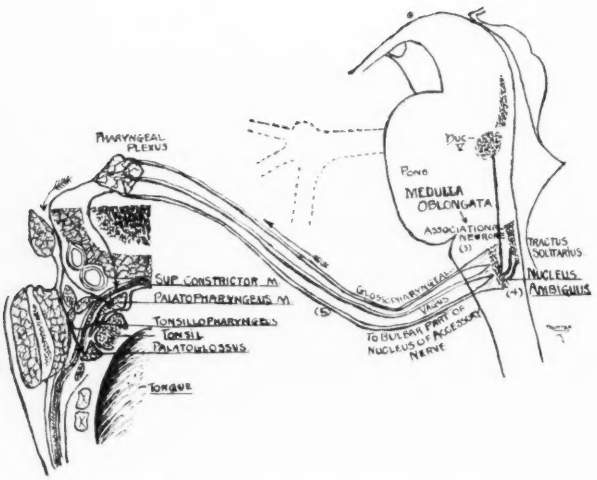


Fig. 4. Efferent nuclei, spinal accessory nerve, showing distribution to muscular attachments of the tonsil.

# Abstracts of Current Articles

Evidencing the quickened desire of otolaryngologists in the Latin countries to withstand the increasing prestige of English and German work in this field, the Societas Oto-Rhino-Laryngologica Latina has been established to carry on the task begun in 1927 by the Federation of the Latin Medical Press. Under the honorary presidency of Prof. Tapia, this year's meeting will be held in Madrid in November. Topics set for discussion include otitic septicemia and sinus thrombosis; mechanism and reactions of labyrinthine stimulation; and cancer of the larynx.

Like the Spanish expositions held in this year, propaganda for this new society is obviously directed toward Latin America. Belgium, Spain, France, Italy, Portugal, Roumania and Switzerland are already represented by delegates of prominence. Membership is limited to those of Latin birth or residence, and only the romance languages may be used for papers or discussions. F.

**The Question of Official Languages at the Next International Congress in Madrid.**

Recent French and Spanish journals give considerable space to rather acrid letters which have passed between Professor Schmiegelow, the diplomatic and urbane presiding officer of the recent Copenhagen Congress, and Professor V. Grazz, of Florence. The latter was successful in forcing the use of Italian upon his colleagues, although English, French and German were previously agreed upon. Obviously few delegates save the Italians got anything out of their contributions.

Professor Tapia of Madrid, who heads the next Congress, agrees with Prof. Schmiegelow that it will be foolish to admit other languages; if Italian is to be included, Prof. Tapia and his colleagues will with equal propriety ask that Spanish be made official. He states proudly that every Spanish otolaryngologist can make use of at least one of the three official languages. This is of course also true of Italy; but it is apparent that feelings of national pride rather than of scienti-

fic expediency are mixed up with the Italian attitude. Certainly not many otolaryngologists from the United States or England would care to visit another polyglot Congress. Many of us would prefer the language of our confrères of South America and Spain. F.

**Primary Suture of Mastoid Wounds (Considerazioni cliniche, ecc.)**

*Prof. Umberto Luigi Torrini (Florence). Arch. Ital. Otol.* 39:547, Sept., 1928.

The author states his dissatisfaction with primary suture of mastoid wounds, having found it necessary to reopen the wound in several cases. He considers it technically impossible to remove every vestige of infective matter from the mastoid wound at operation. These observations occur in a long report on the results of 500 mastoids in the Florence clinic headed by Torrini. F.

**Epistaxis at Various Ages. (Le epistass: nella varie fasi della crescenza.)**

*Dr. G. Calogero (Naples). Arch. Ital. Otol.* 39:611, Oct., 1928.

The author has analyzed many thousands of cases in de Carli's Clinic at Naples, deducing that epistaxis is more frequent at the time of life when greatest growth in height occurs. It becomes rare after maturity; is unusual at the menopause and very rare in old age. Nosebleed is more frequent in males, but does not, as might be expected from the vicarious turbinal congestion often seen, either precede or follow the feminine menstrual cycle. Instead, in both men and women, epistaxis is most frequent when puberty has become well established. F.

**Peripheral Facial Paralysis. (La paralysie Faciale Périphérique.)**

*A. Sargnon and P. Bertein. Arch. Int. Lar.* 8:5-101, Jan., 1929.

This exhaustive monograph presents numerous interesting speculations regarding the possibility of central ganglionic origin for many of the facial paralyses hitherto attributed to cold and exposure. In geniculate ganglion paralysis, herpes zoster oticus may be so transitory as to evade the observer. The authors warn against early attempts to restore function by operation on the nerve; return to function after six months of complete reaction of degeneration may be observed. In the

meantime, tarsorrhaphy and plastic or prosthetic lifting of the corner of the mouth should be done, if necessary, although upward massage is often sufficient to hold up the sagging tissues.

Careful analysis of the facial in its functions as a mixed nerve, recent anatomic and clinical studies, and a very recent and complete bibliography complete this admirable piece of work. F.

**Methods of Examination of the Vestibular Organ. (Les méthodes d'examen de l'organe vestibulaire.)**

*Prof. E. Quix (Utrecht). Arch. Int. Lar.* 8:133-209, Feb., 1929.

The brilliant series of lectures and demonstrations given by Prof. Quix in the LeMaitre course at Paris, in the summer of 1928, are collected here with a wealth of tables and illustrations. The author's theories respecting the function of the otoliths in maintaining static equilibrium are beautifully demonstrated, and the entire contribution must be studied by those who desire light on the interrelation between otolith and canal innervation in the etiology of nystagmus. F.

**Frequency of Bilateral Chronic Maxillary Sinusitis (Die Häufigkeit der doppelseitigen chronischen Kieferhöhlenentzündung).**

*Prof. Dr. Tonndorf (Göttingen), Ztschr. f. Hals, Nasen und Ohrenh.*, 22:1:1, June, 1928.

Examining his statistics for the past three years, Tonndorf was struck by the fact that most of the ethmoid suppurations were secondary to maxillary involvement. Only 5 per cent of the maxillary cases were of dental origin. Several young children were found in the series—one had undergone a year's treatment in a tuberculosis sanitorium and was found to have double polypoid antral suppuration. Of 236 maxillary cases, 143 were bilateral, 93 unilateral; a proportion of 3 to 2. He found practically no cases of isolated ethmoiditis or sphenoiditis. F.

**Healing of the Radical Antrum Cavity (Beitrag zur Ausheilung der nach Luc-Caldwell operierten Kieferhöhle).**

*Prof. Dr. Tonndorf (Göttingen), Ztschr. f. Hals, Nasen und Ohrenheilk.*, 22:1:54, June, 1928.

An interesting confirmation of recent experimental work in Mosher's clinic is afforded by Tonndorf's detailed report of

the histologic examination of both antrums from a patient radically operated upon four weeks previously. Death occurred from abscess of the liver. Both antra were opened through the orbits. The mouth wounds were solidly healed. The large subturbinal openings were smoothly lined, although no mucosal flap had been made. Both cavities were completely covered by a thin layer of fine granulation tissue. Several portions of the wall were excised *in toto*, including the floor of the artificial opening under the inferior turbinate. A thin layer of ciliated epithelium was found extending in over the granulation tissue. The cells which had pushed farthest inward were still of the pavement type. Groups of goblet cells were found in the newformed lining.

In another place, far removed from the nasal mucosa, an island of cuboidal epithelium was found, apparently escaped from curettement.

Between the granulation tissue and the old, red-stained bony wall a thin lay of blue-staining osseous tissue had begun. This is explanatory of the reduced lumen of radically operated cavities, so often seen after healing is complete. F.

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## THE AMERICAN BOARD OF OTOLARYNGOLOGY.

The next examination of the American Board of Otolaryngology will be given on Monday, October 21st, in Philadelphia, preceding the opening of the meeting of the American Academy of Ophthalmology and Otolaryngology in Atlantic City.

Prospective candidates for certificates should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Bldg., Omaha, Nebraska, for proper application blanks.

H. P. Mosher, M. D., President.
W. P. Wherry, M. D., Secretary.

# Books Received

**The Nose, Throat and Ear and Their Diseases.**

In original contributions by American and European authors. Edited by Chevalier Jackson, M. D., Professor of Bronchoscopy and Esophagoscopy in the University of Pennsylvania, in the Jefferson Medical College, and in the Graduate School, University of Pennsylvania, and George M. Coates, M. D., Professor of Otology, Graduate School, University of Pennsylvania. Assisted by Chevalier L. Jackson, M. D. Cloth. 8vo of 1177 pages with 657 illustrations and 27 inserts in colors. Philadelphia and London: W. B. Saunders Company, 1929. Price, $13.00 net.

This massive volume is in reality a system of otolaryngology. It is a succession of essays on the many phases of the subject by men peculiarly fitted for the preparation of their respective chapters. For this reason it is especially valuable as a reference book for laryngologists rather than a textbook for medical students. The material in most cases is unusually well handled, although this work exhibits the shortcoming common to collected writings of many authors: there are hiatuses in the subject matter. The book is, without doubt, the most comprehensive collection of otolaryngological material under a single cover.

**The American Board of Otolaryngology 1929 Directory.**

Volume 3, 8vo of 317 pages. Published by The American Board of Otolaryngology, 1500 Medical Arts Building, Omaha, Nebraska.

A Directory listing the names of 1423 otolaryngologists of the United States and Canada, who have personally appeared before the Board and have been granted a certificate of competency.

**Tonsils and Adenoids.**

*By Irwin Moore, M. B., C. M. (Edin.)*, Late Honorary Surgeon to the London Throat Hospital. Cloth. 12mo of 395 pages, illustrated. St. Louis: C. V. Mosby Company, 1928. Price, $6.50.

A complete and comprehensive work, distinguished especially by its extensive list of references, the material succinctly presented.

The index is commendable for its completeness. A book is no better than its index. Publishers of medical works have

not been awake to this fact. The most thumbed reference books on the shelves are those in which the subject matter is most readily available. Moore's book will be one of these.

**Diseases of the Thyroid Gland.**

*By Arthur E. Hertzler, M. D.*, Surgeon to the Halstead Hospital, Halstead, Kansas. Cloth. 8vo of 286 pages with 159 illustrations. St. Louis: The C. V. Mosby Company, 1929. Price, $7.50.

The first edition of this work was published in 1922. It embodies all angles of the thyroid problem. The first half of the book occupies itself with the morphology and pathology of the thyroid, and is profusely illustrated with photomicrographs.

**The Thyroglossal Tract (Le Tractus Thyréoglosse).**

*By G. Rémy Néris.* Paper. 8vo of 180 pages with 20 illustrations. Paris: G. Doin & Cie., 1929. Price, 30 francs.

For those who read French—a fit companion to the foregoing.

**Your Nose, Throat and Ears.**

*By L. W. Oaks, M. D., and H. G. Merrill, M. D.* Cloth. 8vo of 167 pages, illustrated. New York and London: D. Appleton and Company, 1929. Price, $1.50 net.

A readable little book for the laity,—very simple and elementary, but the authors have obviously gone to great pains to make no misstatements, and to present the material in such a way as not to be misunderstood. It is in no sense a "doctor book," but a clear exposition of the A B C's of the ears and the upper respiratory tract.

**Manual of Diseases of Nose, Throat and Ear.**

*By E. B. Gleason, M. D., LL. D.*, Professor of Otology, Graduate School of the University of Pennsylvania. Cloth. 12mo of 617 pages with 262 illustrations. Philadelphia and London: W. B. Saunders Company, Sixth Edition, 1929. Price, $4.50 net.

The sixth edition of Gleason's Manual,—a convenient handbook, sufficiently complete to be of use to the student or general practitioner. There is nothing particularly noteworthy about this volume. A good bibliography would much increase its usefulness.

# Society Proceedings

## CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

*Meeting of Monday, February 4, 1929.*

DR. EDWIN MCGINNIS, THE PRESIDENT, IN THE CHAIR.

PRESENTATION OF CASES.

**"Papillomata of the Larynx."**

DR. THOMAS C. GALLOWAY presented a case of papillomata of the larynx in a nine-year-old boy. The case had been diagnosed laryngeal diphtheria and the patient was in urgent dyspnea when seen. An emergency tracheotomy was done. He had previously had three operations and twelve to fifteen radium treatments and had not spoken above a whisper for eleven months. Treatment for papillomata had included expectant, with tracheotomy and intubation; radium, avulsion, excision and diathermy as reported by Galbraith and Hubbard. Three treatments were given with the biterminal coagulation and dissection current, under suspension. There was a marked late recurrence in the tracheotomy wound and this was removed through the old wound, working under vision through a bronchoscope through the larynx, and the base coagulated. He found with a relatively small current and a needle electrode, a rather amazing precision possible in the larynx. He believed all of the papillomata were gone and after two months the boy had good cords and a good voice.

Another case demonstrated was an apparently intrinsic carcinoma of the larynx treated by electrocoagulation after laryngofissure. Dr. Cottle had used the tissue cutting current to go through the midline soft tissue and most of the cartilage, and a superficial coagulating and dessicating current apparently destroyed the growth which involved the anterior commissure and part of each cord. There was almost no reaction and in six days the patient swallowed normally. So far the patient had a normal airway and could be expected to have a fair voice.

DISCUSSION.

Dr. Samuel Salinger spoke against the use of radium in treating papilloma of the larynx. In two cases of which he had knowledge the radium had no effect on the growths, but produced adhesions between the skin, the underlying cartilage and perichondrium, and an atresia of the trachea. He knew of no case that radium had benefited in the least. Electrocoagulation of carcinoma seemed to be a beautiful and ideal method, but only time could tell whether all of the growth was actually destroyed, and whether there will be secondary contractures from scar tissue. If the carcinoma is destroyed permanently and an airway can be maintained the method may prove superior to a cutting operation.

Dr. Edwin McGinnis said that in 1914 he treated papillomata by fulguration. He tried it with fair results, but was so impressed with Dr. Lynch's method that he forgot all about the fulguration. He recently had a case similar to Dr. Galloway's, in which he had operated repeatedly for recurrences, some of which were on the epiglottis. Finally he used diathermy under ether anesthesia, removing the ether from the room, and got a smooth end result, without any evident scars and very little destruction.

He also had had some experience with papillomatous formation around a tracheotomy wound, and removal of the tracheotomy tube with the healing of the wound pinched off the growths. He had treated one case in this way and the patient was able to breathe through the larynx, and there was little scarring. He had followed New's procedure in two cases, and was greatly pleased with the way in which they healed with so slight reaction. In twenty-one days one patient was healed and out of the hospital.

The first cases were operated under ethylene gas anesthesia by putting in an airway through the trachea and using the Mackenty tubes.

Dr. Harry L. Pollock said they had tried surgical diathermy but had discarded it and resorted to surgical measures entirely. The more experience one has with laryngectomy the easier it becomes and the better are the results. In intrinsic carcinoma this operation will cure the patient, and he

believed it to be the best procedure. In a case in which the growth was located on one side they did a laryngofissure and used diathermy a year and a half ago and the patient was still well, but the growth was very small. In an operation performed that morning he used the Gwathmey synergistic anesthesia. The patient made no compaint and was in good condition at the end of the operation. He thought this method worked better than a general anesthetic, particularly unless one used the Mackenty tubes, for if the patients are awake they can expectorate the mucus and dangerous complications can be avoided.

DR. GALLOWAY, closing, thought it should be remembered that there is little scarring following electrocoagulation, perhaps less than after any other operation. They had not a large number of laryngeal cases, but from their experience with coagulation in conditions about the head, were convinced it offered much. Local anesthesia, blocking the superior laryngeal nerve after Braun's method, had been very satisfactory.

DR. OSCAR BREITENBACH, Waukegan, Illinois, presented his inaugural thesis, entitled .

**"The Pathologic Significance of Deviated Nasal Septa."**

(AUTHOR'S ABSTRACT)

The records of large hospitals show that next to tonsillectomy, the submucous resection of the septum is now the most commonly performed operation in otolaryngology. A definite field for prophylactic achievement presents itself in connection with the many factors undermining normal respiratory function of nasal air passages. The conservation of function, and the restoration of function, can be accomplished by the removal of pathology secondary to deviated nasal septa. The apologetic attitude of many in detailing tonsil and septal pathology is unnecessary. No subject in otolaryngology is worthy of greater scrutiny and more study than that of septal deviation, especially the evaluation of its pathology as a definite disease factor.

The deviated septum, simulating in the minds of many an elementary subject in the practice of otolaryngology, has fos-

tered timidity and many seem hesitant to approach this subject, because of a possible odium of mediocrity.

The cry has been raised both in and outside the ranks of our specialty that too many septa are being operated on unnecessarily. Many septum operations have been performed without well supported grounds, but considering the varied pathology that a deflected septum may originate, it is not altogether improbable that many deflected septa are not operated on that should be. Statistics from both European and American sources indicate that over 75 per cent of all adults have deflected septa. They are either of developmental or traumatic origin, the former preponderating. Septa are much more commonly deviated to the left than to the right, and ballottement is to be remembered as a factor in deviation.

According to the location, the septal deflection may block the air stream to the lung, to the sinuses, or to the eustachian tube and the middle ear. The pathology of the respiratory tract and its very definite relation to deviated nasal septa must be kept in mind. Because of the prevalence of deflected septa, special care must be taken to rule out other possible causes of the patient's complaint. Disappointment following submucous resection will occur less often if this is done. Slight deviation that is not a factor in aberrant function should be strictly left alone. Air hunger from general conditions, such as anemia, pulmonary disease, and myocardial affection, should be kept in mind.

A plea for postoperative data in the general field of otolaryngology is made.

DR. E. W. GARDNER presented his inaugural thesis, entitled

**"Acute Suppurative Mastoiditis with Purulent Leptomeningitis. (Hemolytic Streptococci.)"**

The patient, Malcomb, aged 7, was one of twin boys, both of whom were operated upon the same day for purulent mastoiditis. The other twin recovered uneventfully. Both boys had had frequent attacks of purulent otitis media since childhood. In 1924 both boys had hemolytic streptococcic otitis media, bilateral. Courtney recovered following paracentesis. Malcomb had bilateral paracentesis, but required later mastoidectomy, unilateral. During the following three years

(1924-27) both boys had recurrent purulent otitis with paracentesis and recovery.

March 4, 1927, Malcomb presented unilateral purulent otitis. Temperature 104° F. Drum incised. No culture taken. The following week the entire family were ill with epidemic upper respiratory infection and patient was seen (as the family lived in a suburb) by the family physician. The patient was again seen eleven days after paracentesis. Classical mastoid symptoms were presented—deafness, canal sagging, temperature 100° F. WBC 10,500, polys 74, tenderness over and below mastoid. There was some neck tenderness, but no suspicion of meningitis at that time. Skiagrams were positive. There was no postauricular swelling. Operation showed a necrotic mastoid bone and the antrum filled with necrotic granulations. There was no demonstrable dehiscence of inner table.

The following day morning temperature was normal. At 4:00 p. m. it was 101.2° F. and continued between 100° and 106.8° F. until the patient's death on the 16th day subsequent to operation, the 27th day subsequent to paracentesis, and 34th day subsequent to first respiratory symptoms.

The following are the points of especial interest: (1) Positive meningitis symptoms first day subsequent to operation. (2) Operative wound remained clean, but showed no reparative process. (3) WBC ranged from 10,500 to 42,900. Polys from 79 to 98 per cent. (4) Daily spinal puncture with removal of 5 to 20 cc. fluid. (5) Spinal fluid cell count began at 1,100 and advanced to 9,000. Hemolytic streptococci. (6) Blood culture was always negative. (7) Skiagram of head indicated brain abscess posterior to operative area. Disproved at postmortem. (8) Spinal mercury manometer showed lessened permeability of left lateral sinus (by Dr. Lewis J. Pollock). (9) The patient received, in all, 112 cc. blood serum, by subcutaneous injection, from his mother (who had been ill with infection identical, so far as could be ascertained, to that of patient and whose blood, it was hoped, would be rich in antibodies beneficial to the lad). These injections produced reaction in the lad (increased temperature, once a chill, etc.), but no definite conclusion as to their value could be drawn. (10) Dr. Loyal Davis made an exploration

of the brain with a needle for left temporal abscess. (Necrotic tissue, but not free pus, was found at postmortem.) (11) Edema of the lids, and the fundi vessels were engorged, suggesting there was cavernous sinus thrombosis, which was not found at postmortem and was probably due to the great intraventricular pressure as found at postmortem.

Postmortem findings: (By Dr. L. E. Hines, pathologist, St. Joseph's Hospital.) Clot in left lateral sinus, adherent to wall but not occluding. Surgical defect shows no opening through the inner cranial table. The blood vessels of brain are engorged and everywhere there is evidence of brain tissue compression. In sulci along the blood vessels is a yellow purulent exudate, especially over the left hemisphere. Lateral ventricles are markedly dilated, especially the left, and linings covered with purulent-like exudate. The left ventricle extends posteriorly to within 1 cm. of posterior surface. (It probably was this extension which, in the X-ray, was taken to be an abscess.)

Anatomical Diagnosis: (1) Generalized suppurative leptomeningitis. (2) Marked dilation of lateral ventricles, especially left of brain. (3) Left suppurative mastoiditis. (4) Slight thrombosis, left lateral sinus. (5) Marked generalized pallor and emaciation. (6) Cloudy swelling of all viscera. (7) Acute splenic tumor. (8) Petechial hemorrhages of lining of small intestine.

One year subsequent to this, the surviving lad required mastoid drainage of the previously unoperated ear. He recovered uneventfully. The organism again was hemolytic streptococcus.

During his illness his mother said she had had a slight sore throat for several weeks. Examination showed subacute infection of tonsil stumps. Culture showed hemolytic streptococci identical in all respects to the streptococci found in the patient's cultures. She had a tonsil operation several years prior to the birth of her boys, and had had no acute tonsillitis at any time since then. It was her habit to kiss the boys directly upon the mouth. Her tonsil stumps were removed, since which time Courtney has had no recurrence of otitis media.

Drs. Wm. Burmeister, Wasko, Lewis J. Pollock, A. A. Hayden, Loyal Davis, L. E. Hines were called in consultation.

DR. E. W. HAGENS, collaborating with DR. G. E. SHAMBAUGH, presented his inaugural thesis, entitled

**"Malignancy of the Middle Ear, Bilateral, With Autopsy Findings."**

(AUTHOR'S ABSTRACT)

The patient sought medical aid on account of stuffiness in the left nostril and this case is of interest because the patient was observed over a period of six years. Tissue was removed from the left side of the nose and proved to be epithelioma. Radium was applied and the tumor disappeared. After several years the patient noticed tinnitus in the left ear, and examination revealed a tumor mass in the upper part of the cavum tympani, shining through the drum membrane. Radium application did not affect the tumor development. The hearing tests showed mainly a defect for the upper part of the tone scale on that side, and practically normal reactions on the right side. The vestibular mechanism was intact.

Later, stuffiness recurred on each side of the nose. Tumor tissue was seen on both sides in the ethmoid region, and sections were again made from the left side. Dr. LeCount reported epithelioma. The tumor tissue disappeared from the nose a second time on the application of radium. The patient next noticed fullness in the right ear and tumor tissue was seen shining through the lower portion of the drum membrane. From this time on until her death (from another cause) she complained of increasing stuffiness of the ears, tinnitus, deafness, attacks of vertigo, and dryness of the nose. Facial paralysis on the left side was noted but the patient did not complain of it. The hearing tests showed gradual increase in deafness on both sides, especially the left, until very little was heard in this ear. The vestibular tests gave increasingly diminishing responses.

The gross examination of the right temporal bone did not show any outward evidence of tumor, but on the left side several masses of tumor tissue had broken through the tegmen tympani, and what was left of the middle ear cavity was filled in by tumor masses.

Microscopic examination of the right temporal bone revealed extensive tumor invasion of the cavum tympani up to the level of the epitympanic space. There was slight invasion of the bony capsule about the cochlea. For the rest, and particularly the cochlea and vestibular end organs, no tumor invasion was noted. The sections from the left temporal bone showed very extensive infiltration of the bony labyrinth, and extension of the tumor into the utricle, the cristae of the canals and the cochlear duct at its beginning in the vestibule. The saccule and most of the cochlea were free from the invasion. The seventh nerve region was replaced by tumor masses.

The tumor was fairly vascular and gave a papillary appearance under the microscope. The sections from the nose appeared similar to those from each ear. Those from the ear appeared most like that of a basal cell carcinoma. It is probable that the tumor extended by direct growth from the nose backward and outward into each ear. The article is accompanied by a number of microphotographs.

DR. FRANCIS L. LEDERER presented his inaugural thesis, entitled

**"Prosthetic Aids in Reconstructive Surgery About the Head."**

(AUTHOR'S ABSTRACT)

I wish to consider only those cases of the true reconstructive type, namely, those in which there is a total or subtotal loss of parts.

Syphilitic nasal destruction is usually classified in three degrees, (1) the saddle nose, in which the saddle is confined to the bridge; (2) a nose in which the tip is retracted upward or to one side, due to loss of mucous membrane as well as of the bony and cartilaginous support, and (3) destruction, partial or total, of all the nose (lining, structure and skin).

The cosmetic reconstruction of first degree, and in most instances of second degree, syphilitic nasal deformities, has on the whole yielded satisfactorily to the variety of technics employed today. The repair of third degree syphilitic destruction of the nose has been more difficult than deformities due to other causes, because of the devitalization produced in the

area attacked and also because of the disturbed circulation in neighboring tissue. Covering the parts with skin is an improvement over the original deformity, but the success of such endeavors is apt to be over estimated by the operator without considering the psychology of the patient.

Success in reconstructive cases has been only temporary, and too often the result at the end of surgical endeavor was deformity replaced by monstrosity.

I have been disappointed in the net results of my painstaking endeavors and in a measure feel guilty for having subjected my patients to operations which not only entailed a great economic loss and suffering on their part, but in many instances jeopardized their health and lives.

Both the congenital and acquired type of complete or partial absence of the auricle has occupied my attention for a number of years. Careful and painstaking attention has been given a rather large group of these cases, and I found that the result of this labor has been most discouraging. Van Dijk comments on the absence in the literature of reports of cases in which the entire auricle has been reconstructed by surgical measures. I have only to refer him to the work of Beck who, on innumerable occasions, has published cases embodying work of supplanting an entire auricle. Van Dijk draws the conclusion that the loss of an entire ear seldom occurs. He also states that the aural appendage is not cosmetically essential in women, as they are able to hide their deformity completely under their hair. However, I find that regardless of sex, the desire for replacement of the parts is true in practically every instance. A few years ago Beck described clearly the need of correcting deformities because of certain vital psychologic factors, which were so important to keep the person in his or her social sphere.

Van Dijk, at the end of the series of operative procedures, was not satisfied with the final result. He found the ear too bulky, not sufficiently erected and the fossa scaphoidea and the antihelix not built up, and he planned in his next case to get the ear more erect through implants of cartilage, giving it more of a shell-like appearance. This supports my conclusions that in a large number of cases in which I had an opportunity in each instance to improve my technic, I

could not shape an ear to resemble a normal ear, but replaced it by means of a bulk of misshaped tissue.

I am indeed indebted to my former association with Dr. Joseph Beck, for having given me an impetus, as well as in a large measure for having furnished the clinical material for this work and for the splendid cooperation I have had at all times.

For a number of years, during which total deficiencies of the nose and ear also were corrected by surgical measures, certain physiologic, anatomic and economic factors often presented which made it absolutely necessary to use some form of prosthesis in these cases in the form either of hard rubber or of some light metal. I found these objectionable for many reasons, viz., the cost of manufacture was high (they were of such material as was easily broken or bent and would frequently have to be made over); the color to match the neighboring skin was difficult to obtain, and if finally it did match, the color would come off; the prosthesis would in most instances have to be fastened by a heavy spectacle frame; and, lastly, but of great importance, the replaced part always had a stiff, artificial appearance that attracted as much attention as did the original deformity.

When I went to Paris, I was struck by an observation made in a theater of international fame in which dolls of a flexible rubber material were being sold. It occurred to me then that the material appeared so elastic as to be invaluable in prosthesis. Much to my gratification, I found that at the University of Berlin a similar type of material was being utilized in the manufacture of face prostheses. Credit must be given the late Professor Adolph Passow, who, at the suggestion of Warnekros, in 1913, induced his laboratory technician, Klocke, to work out such a method of producing artificial ears and noses. The procedure has been perfected by Klocke, and I feel sure that these prostheses comply with all the demands which can be made on such substitutes. They are not expensive, are easily made and possess a desirable elasticity and durability. I employ a dental laboratory technician who takes care of most of the time-consuming detail work, and it is recommended that one who has experience in doing prosthetic work should work in conjunction with the

physician. No doubt as this method becomes better known, there will be modifications on modifications. I am constantly working to attain an even more satisfactory material than I have at present, and perhaps in the future a perfect formula will be attained.

It is far more difficult to replace by prosthesis parts of ears, for example, than to replace the part *in toto,* but it has been my experience that these patients will not consent to having any remaining part removed by surgical procedures. It is therefore necessary, in many instances, to cover useless lobules and folds of skin that could well have been eliminated. In such cases the prosthesis has not been made without its critical points, but at least the patient's desires have been complied with. While mechanical appliances will not take the place of nature, yet by this method many persons can be relieved of untold embarrassment, suffering and economic loss.

# CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

*Meeting of Monday, March 4, 1929.*

DR. EDWIN MCGINNIS, THE PRESIDENT, IN THE CHAIR.

## CASE REPORTS.

### "Carcinoma of the Larynx. (Presentation of Specimen.)"

DR. T. C. GALLOWAY qualified his enthusiasm of the preceding meeting in regard to the treatment of carcinoma of the larynx, and stated that the patient whose case was reported at that time developed a follicular tonsillitis, followed by bronchopneumonia and death. Postmortem examination revealed a carcinomatous involvement of the left side of the larynx which was inoperable, even by laryngectomy. The patient had shown no evidence of recurrence during life, but at necropsy a large mass was found below the epiglottis which was definitely malignant and some other material from the larynx was doubtful.

One thing in favor of the method was that there was no denuded cartilage left, there was very little destruction and the area had healed with a rather smooth scar throughout, but the case was disappointing and he felt that he had overestimated the value of the method. Replying to a question he stated that they had not split the hyoid, but he felt that it should be done in such an extensive case.

## DISCUSSION.

DR. G. W. BOOT reported a case under his care which apparently was limited to the right vocal cord. He split the larynx with the radio knife, and found outside in the median line at the cricothyroid junction a growth which looked like a gland, but which microscopic examination showed to be carcinoma.

DR. ROBERT H. GOOD, referring to a case of carcinoma of the vocal cord, now in the hospital, stated that this case was identical to the pictures Dr. Mackenty showed at the last meeting. A growth was removed from her right vocal cord and histologic examination revealed a papilloma with indica-

tions of carcinoma, as reported by two laboratories. Dr. Mackenty said if two laboratories reported carcinoma the patient should be operated upon. He observed the patient for six weeks after removal of the papilloma and could see three nodules appear on the cord which grew rapidly. He then decided to destroy the right vocal cord with diathermy, using a Heslinger pharyngoscope. The patient was able to breathe well following operation, but a few weeks later he found a recurrence in the thyroid cartilage, and then decided to do a laryngectomy. Examination of the specimen removed showed involvement of the entire right side of the thyroid. The patient did well following laryngectomy, but the use of diathermy at the first operation did not prevent the recurrence of the growth.

Dr. Joseph C. Beck expressed the opinion that in most cases, the use of diathermy was simply a laryngectomy without taking away the larynx, and that if the cartilage is not completely destroyed a recurrence may not be prevented. Two years ago he performed a laryngofissure for a patient about forty years of age and destroyed a big papilloma, which was suspected of being carcinoma, by means of diathermy, but he was not sure that it was not a case of pachydermia of the larynx. He complimented Dr. Galloway on the specimen presented, because it added another truth.

Referring to the case reported by Dr. Boot, he said it was common to find a gland at the junction of the thyroid and cricoid cartilages before one opened the larynx. However, it was not always malignant.

Dr. G. W. Boot said that a papilloma in an individual past forty is almost invariably carcinoma. If this growth is destroyed by diathermy one does not destroy the patient with pneumonia, as so often happens with a laryngectomy.

Dr. Alfred Lewy stated that in the case he observed with Dr. Galloway the unoperated area of the mucosa was perfectly smooth at the time of operation, and there was no palpable gland so far as their diagnostic skill could discover. He thought their case proved that it is impossible to find all of the carcinomatous involvement. He still considered

diathermy a good method for a strictly intrinsic growth, but thought it difficult to prove which cases should be handled in this way.

Dr. Edwin McGinnis reported that a patient on whom he had done a total laryngectomy had returned with a recurernce in the glands of the neck. He believed if Dr. Galloway and Dr. Lewy had done a laryngectomy they would probably have missed the carcinomatous glands in their case.

Dr. George W. Boot reported the case of a young Mexican boy whom he had seen recently, who had a fight with another Mexican, who shot him. One bullet went into the left nares, through the septum and lodged in the right sphenoid sinus, and was removed through the right nares. Skiagrams were presented.

Dr. Walter Theobald presented a paper, entitled

**"Outlining the Maxillary Sinus by Injection of Lipiodol Under Pressure."**

(ABSTRACT.)

The object of this paper is to present the method and results of filling the maxillary antrum with opaque solutions for the purpose of distinctly outlining gross hyperplasias, polyps, cysts and malignancies, and does not include the sphenoid, frontal and ethmoid sinuses.

The method used by the writer consists of a complete air displacement of the antrum by injecting lipiodol with a syringe, under pressure. After cocainizing the middle meatus a canula with a filled syringe is introduced by way of the natural or accessory opening into the antrum. There is always enough room around the canula for the escape of air and injected fluid. The head is then tipped to the side so that the vertical anterior posterior plane of the head becomes horizontal, or parallel to the floor, thereby causing the affected antrum to be the dependent one, and its natural ostium uppermost. In this position a mixture of equal parts of lipiodol and olive oil is slowly injected to the point of overflow, from 3 c.c. to 7 or 8 c.c. sufficing. The patient is not allowed to change the position of the head until two X-ray views are taken, an antero-

posterior view with the X-ray beam placed horizontally, and a lateral view taken from above with the vertical beam. The stereoscopic plates do not prove always of great value. When injection by way of the natural ostium fails a trocar introduced into the inferior meatus, with proper fittings for the syringe, may be used, but the other procedure is successful in 85 to 90 per cent of the cases. Care should be taken to have all connections fitted perfectly, so that no oil will escape and the antrum will become positively filled by pressure. No untoward effect has been observed from the use of lipiodol. The simple procedure of irrigating the antrum after the opaque solution is used obviates possible complications.

This procedure visualizes the extent to which an antrum may be completely or incompletely filled. The thickness of the mucosa is at once noted by measuring the distance from the bony outline of the cavity to the outline of the opaque fluid. It may appear as a simple thickening of the mucosa, or show an irregular outline pictured as indentations caused by polypi and granulation tissue. The results have been very satisfactory when two different views are taken, and the conclusions in a number of cases examined have been substantiated by the microscopic findings at the time of operation. Several cases are reported.

DISCUSSION.

Dr. Joseph C. Beck said he had followed the work of Dr. Theobald and was much impressed with the proof that filling defects can be shown well with opaque mediums. Several years ago Dr. Beck experimented with bismuth and with iodin ointment or pastes by injection into the antrum and other cavities, but found this was not a good thing to do for he could not wash out the antrum afterward and make the studies he wished to make. He has not used lipiodol extensively and was not particularly enthusiastic about its value. He thought one could visualize what is going on in the cavities from an x-ray and clinical examination and on operation will sometimes find a polypoid condition in the antrum. Dr. Theobald's experience with many cases and his specimens proved that this condition occurs more often than is thought.

Dr. Beck believed the question of removing the mucous membrane from the antral cavity entirely to allow regeneration of the membrane remains to be proved. It has been demonstrated experimentally on dogs but not on humans and he was skeptical of its value.

He called attention to the recent work of Dr. Hickey of Ann Arbor in bringing out tissue details by long distance radiography. Dr. Hickey places the patient sixty to ninety inches from the tube and gives a much longer exposure than is usually given at contact photography, the time depending upon whether he wishes to bring out details of soft tissues or more dense ones. The method is very successful in showing the details within the thorax and if it could be utilized for work about the head it would be superior to any kind of injection for demonstrating a filling defect. He believed, however, that much more will be heard of lipiodol, as work with it is just beginning.

Dr. John A. Cavanaugh stated that in 1920 he read a paper at a meeting of the Illinois State Medical Society on the injection of barium in buttermilk and later the use of barium in malted milk. Since then he has done a good deal of work with lipiodol and has also continued the use of barium in malted milk, which he thought gave better detail and outlines and was not so subject to fallacious interpretation as the results from lipiodol. In a case injected recently, after introducing the needle serous material kept flowing from the canula so he introduced lipiodol under pressure. This gave a picture of the maxillary sinus filled with small globules. He then cleaned this out and injected with barium in malted milk and a much better definition was obtained. He was not sure as to the value of lipiodol injections, yet he felt continued perseverance along this line might uncover something worth while.

Dr. A. A. Hayden expressed the opinion that it made no difference how far the light is from the sinus, but that it made all the difference in the world how far the X-ray plate is from the sinus. A light across the room will give as clear a reflection as if it were nearer.

DR. EDWIN MCGINNIS said that at a recent clinic in Boston one of the clinicians in filling the antrum plugged up the opening with cotton and then injected the lipiodol under pressure.

He cited the case of a patient he had under observation, whose physicians had been searching for local infection. Her tonsils were removed and she was then referred to an oral surgeon. He pulled an upper molar and curetted the cavity, and got into what he thought was the antrum, which he treated by irrigation and currettage. The patient did not improve and was referred back to the oral surgeon. She finally got to the surgical department, where they passed a catheter, filled the cavity with lipiodol, and this brought out the fact that she had a large misplaced ethmoid cell.

DR. WALTER H. THEOBALD (closing), approved what Dr. Beck said about operating on an antrum and presuming diseased tissue present when the mucous membrane is normal, or nearly so. In some cases he has found the mucous membrane a little thickened but not sufficiently so to justify a radical operation. By means of the opaque substance one can show to his satisfaction when the mucosa is thickened, or when the antrum contains a polyp or foreign growth.

It is not possible to show all of the inner lining of the antrum by means of the endoscope. He has seen the inside of an antrum two to five years after operation and considered the lining to be normal mucous membrane, because no discharge was coming from the antrum. In other words it was believed to be a cured case.

He believed the distance of the light from the patient made no difference. The beam of light is placed always the same distance away from the patient for each exposure.

In the case mentioned by Dr. Cavanaugh the antrum was probably fairly well filled with polypi and the lipiodol was not injected under sufficient pressure to fill the spaces between them correctly. When he injected the second time it showed the crypts between the polypi. In one of his own cases at the time of irrigation a flaky material was washed out that had the appearance of cholesteatoma, and when the picture was taken it showed the antrum nearly filled with a growth.

Dr. Samuel J. Pearlman read a paper on

**"Retropharyngeal Abscess of Otitic Origin."**

(AUTHOR'S ABSTRACT)

Retropharyngeal abscesses of otitic origin are not seen very often. There must be, however, a number of these which go unrecognized. The anatomical routes by which pus from the external and middle ear and from the mastoid reach the retropharyngeal space are devious and complicated. A retropharyngeal abscess may arise from a furuncle of the external canal which invades the parotid space and then spreads to the retropharyngeal space; it may take its source from an extradural abscess of the middle or posterior cranial fossa. It may arise from an otitis media and attack the retropharyngeal lymph nodes by way of the lymphatics. They are all of great interest and should be more carefully studied.

DISCUSSION.

Dr. Edward P. Norcross cited a case which was under his observation five or six years ago. The other symptoms overshadowed a retropharyngeal abscess. It recurred several times, disappearing after a radical mastoid operation was performed.

Dr. John A. Cavanaugh thought that Gruber's idea of the anatomical defects of the Glaserian fissure was very logical and more plausible than many of the other causes given.

Dr. Samuel Salinger said that histologic proof was really the most conclusive in these cases, and Dr. Pearlman had had at least one opportunity of proving the route by which the abscess reached the retropharyngeal space. In the case seen by Dr. Pearlman and himself at postmortem they found that the tip of the petrous bone was entirely necrotic and bathed in pus. The pathologist on inserting his finger in the nasopharynx found a distinct bulging which corresponded to the necrotic area of the petrous bone. Had the patient lived a little longer it would have been possible to determine it clinically. The postmortem made it evident that the necrosis of the tip was working the infection downward toward the postnasal space.

DR. LOUIS OSTROM (Rock Island, Ill.) cited the case of a patient he saw with Dr. G. L. Eyster some years ago for whom he removed a fish-bone from the lower end of the esophagus. Two or three days later the patient complained of sore throat, which developed into a double otitis media and double mastoiditis with complete necrosis of both mastoid tips, with very marked edema of the tissues in the neck which looked as though they would necrose. Profuse discharge, more than could be accounted for, lasted for several days after operation. When a large, long probe was used, it passed into the neck, anterior to the cervical vertebrae, and came out on the other side of the neck through the other mastoid wound. Gauze packing was put in extending through the neck. The patient also had nephritis. He finally returned to his home in Chicago and the ultimate outcome was not known. This infection undoubtedly pursued the retropharyngeal path, as described by Dr. Pearlman.

DR. JOSEPH C. BECK recalled a case that he reported before this Society some twenty-five years ago in which when he irrigated one ear the water came out through the other. That was definitely a case of necrosis extending through one of the routes described by Dr. Pearlman. He also saw a case with Dr. Mundt some five years ago of a paratonsillar abscess complicating mastoiditis. The labyrinth function was destroyed but there appeared to be no connection with the suppurating ear. There was some evidence of an extradural complication. In that case an incision was made by the doctor for a paratonsillar abscess, but on digital examination a retropharyngeal abscess was found. The patient died but no necropsy was obtained to prove the route of the infection. Several years ago Dr. Beck reported a case of Bezold's mastoid and at that time stated that the routes of infection were those described by Cunningham. At present he has a case of retropharyngeal abscess under his care, in a child patient who was brought in from Wisconsin with this very acute condition. On puncturing the abscess the thin chalky white pus spurted out over the patient's face. The patient was running a high temperature, even after operation, which he felt was due to the associated lymph gland involvement, but it might also be due to some organic complication.

**"Phonetics in Relation to Otolaryngology."**

DR. G. OSCAR RUSSELL, Director of Phonetics, at the Ohio State University, by inivitation, addressed the Society on "Phonetics in Relation to Otolaryngology," and demonstrated his autofonofaryngoskop and autoperilaryngoskop.

(AUTHOR'S ABSTRACT)

Let us summarize the reasons for the unusually sharp, and more complete laryngeal view just demonstrated through the perilaryngoskop and fonofaryngoskop, manufactured per my specifications by the E. S. I. Co., Rochester, N. Y.

1. The socalled retracted or fallen epiglottis cannot be manifest during pronunciation of a clear vowel "ee" (i as in peep), for as the tip moves back towards the wall of the pharynx, the quality shades off into an "uh" (ɔ as in cut), or "ah" (a as in calm).

2. Therefore any instrument permitting the subject to pronounce a clear and unhindered "ee" makes it possible for the examiner to inhibit retracted epiglottis interference—provided always that gagging or other extraneous disturbance does not inject itself.

3. This vowel also distends the lower pharynx the most, brings the tip of the epiglottis as far anterior as possible, likewise relaxes the posterior pull on the cushion of the epiglottis and its approximation with the cartilages of Wrisberg.

4. If pitched conveniently high, and the voice quality is free flowing (i. e., not constricted or strident, nasal, or guttural), we get the maximum glottal lip view. Even the anterior commissure is laid bare, and one sees well up under the cushion, getting also a much wider lateral view under the ventricular bands.

5. But a clear unhindered "ee" cannot be produced where the ordinary laryngeal mirror is used, nor with any lens system in a straight tube larger than a pencil in diameter. Hence the cystoscopes, autophonoscopes, bronchoscopes, endoscopes, and other like instruments, fail to give the complete view. For to pronounce this vowel clearly, subjects regularly force the tongue up against the full length of the hard palate, forming a very narrow arched tube through which a view of the back pharynx cannot be had. And if the buccal cavity

is forced open, the vowel changes in quality towards "eh" (ɛ as in pep), "uh" (ɔ as in cut) or "ah" (a as in calm), thus leading at the same time to their subconscious posterior closure of the epiglottis.

6. The perilaryngoskop being but 3 mm. in diameter, avoids all such difficulties, gives a wide-angle view of the interior larynx and surrounding pharynx simultaneously. At the same time it may, if dexterously handled, eliminate the reflex causes of gagging. Hence it permits an examination in subjects heretofore thought impossible.

7. Gagging constricts the ventricular bands; likewise approximates the cushion of the epiglottis and cartilages of Wrisberg. As a reflex, it is Nature's way of keeping foreign substances out of the larynx; hence it is aroused when the interior larynx is touched by any hard body, if extraneous contact is made or force brought to bear on the epiglottis to keep it from closing, or even if the back velum is touched, or pressure brought to bear on the tongue in order to apparently force an entrance into those sensitive precincts.

With either the perilaryngoskop or fonofaryngoskop, these are reduced. The lens of the first makes it possible to get a clear view of the whole interior larynx (since it permits a clear vowel "ee"), when the distal end is even 20 to 30 mm. from the pharyngeal wall. And it need not touch the velum, much less the epiglottis. The front tongue is free to move at will, hence the psychological impression of extraneous force is eliminated.

The fonofaryngoskop is much larger, hence does only the last. But it gives a much brighter image and a view of more of the interior larynx and cords than can be had with an ordinary laryngoscope. And the autofonofaryngoskop has the advantage of permitting doctor, subject, any number of relatives, students, or others in the room (if desirable) to see the view simultaneously.

DISCUSSION.

Dr. Elmer L. Kenyon expressed his appreciation of the valuable work being done by Professor Russell, and pointed out that prior to fifteen years ago laryngologists believed that the vocal cords worked by one mechanism and the mouth by

another. Now it is known that when one produces a vocal tone the muscles of the mouth, neck and the vocal cords act as one unit. Dr. Russell's instruments make it possible to see what occurs in the larynx for each individual sound, the first time this has been possible, and this possibility constitutes an important turning point in the understanding of laryngology, so far as the production of speech sounds is concerned. He was much interested to know that the deafened child and adult could be handled in the way that had been described. Not long before, Dr. Kenyon had spent a day in a state institution for the deaf, where they had all kinds of teaching, but so far as he could find out, took no cognizance of the fact that a child might be able to hear. He felt that through Professor Russell's conception of the employment of residual hearing, the teachers in schools for the deaf could be brought to accept more scientific methods.

Dr. Kenyon has been studying the extrinsic muscles and his work dovetailed almost perfectly with that of Professor Russell's studies of intrinsic laryngeal action. He prophesied long ago that the vocal cords would be found to have a distinct apposition for each of the principles of sound production and this statement is being verified by Professor Russell's studies. In his work to improve the hearing of the deaf he has worked only with those who had no hearing, while Professor Russell has worked with those who retain a slight amount of hearing. Dr. Kenyon has found that a study by the patient of the movements of the thyroid cartilage of the larynx is capable of becoming an effective substitute for the hearing in attempting to guide the speech apparatus to normal voice attainment. If an adult loses his hearing almost inevitably his voice becomes markedly altered but in the way mentioned he had been able to change a very high pitched, strained voice back to normal. He was sure that otolaryngologists would realize the value of the work being done by at least one of the men who work with disorders of speech.

Dr. E. W. Hagens said that in the examination of the school children in the State institutions they found, in the audiometer charts where they used the 2A, the hearing these children had was either below 2,000 vibrations or above and below both. They did not find any children who had a rem-

nant above 2,000 alone. They use two charts which give the relation of hearing in respect to the speech defect. One chart is arranged on the basis of marked speech defect, and one with practically normal speech. They realize that a child may come in with a bad speech defect and improve after a time, so their tables probably had some defects.

In comparing the tuning fork tests with the audiometer it was found that the audiometer picked up some remnants that the tuning forks missed, and he believed the audiometer is more valuable in this way for the remnants can be placed more definitely. In the Pittsburgh school one person said he could feel the 256 and 128, but there was no rubber cap over the piece, which he believed accounted for it. They have found that they cannot diagnose the type of hearing loss from the audiometer records. He believed that Professor Russell's chart added to what they did would be of much aid to the teachers and the children. In some of the schools the teaching was poor, but in some it was very scientific, and they were using all the methods possible and making use of all the hearing the child had. In some institutions they used an apparatus that magnified the voice, whereas previously the children had been taught by the alphabet method. In other schools they insisted upon the oral method entirely. Dr. Hagens believed that with Professor Russell's system it should be possible to teach much more effectively and scientifically.

DR. ALFRED LEWY said that according to Professor Russell's statement it would be simple to produce an instrument that would amplify in any desired pitch, and damp in others. So far as he knew there is no instrument in the American market at present that amplifies higher tones without amplifying the low ones. Such an instrument would be desirable for the cases of inner ear deafness that are not helped by the present telephone apparatus, which is better suited for those patients whose deafness is largely for low tones. Dr. Lewy has made this suggestion to engineers of the Bell Telephone Company, but nothing has come of it. Dr. Flatau presented an instrument before the German Otolaryngologic Congress which it is claimed damps the low tones and amplifies higher ones and is suitable for inner ear deafness. Whether or not this instrument is small enough to be practical he did

not know, and asked if Professor Russell could throw any light on this subject.

DR. ROBERT SONNENSCHEIN expressed his gratitude to Professor Russell for his address and for demonstration of the apparatus. He thought that the speaker was a little pessimistic regarding the functional tests of hearing. So far as testing with speech is concerned, in dealing with individuals with only small remnants of hearing it is very difficult to get a fair idea of the state of the hearing, but in ordinary practice the use of speech is most important, for the criterion of the efficiency of the ear is its ability to understand the human voice. It is practically impossible to standardize the human voice, but if one accustoms himself to using the residual air, and makes the tests in the same room with the same furniture, etc., thus getting the same systems of waves, and the same reflection of sounds, one has a fairly accurate test. The intensity of sound varies inversely to the square of the distance, but this applies to sound generated in the open.

In considering whether a person has marked impairment of hearing, the use of the whispered voice is of great aid. It is very difficult to obtain any pure sound even with a tuning fork. With the average tuning fork the first overtone sound has a frequency about five or six times that of the fundamental. Nevertheless, with the simple expediency of placing weights on the forks, as shown by Bezold, or using rubber bands, one can secure practically a pure tone. If present at all, the overtone will not disturb the patient.

Referring to the duration of vibration of tuning forks, Dr. Sonnenschein said that unless one strikes them very hard there will be only slight variation. If one knows the "constant" of decrement or damping, and strikes the fork in a uniform manner he can, as Fletcher has shown, by means of that "constant" determine the loss of hearing in sensation units just as with the audiometer. While many criticisms may be offered concerning tests of hearing, the principal tests used uniformly will give practical and valuable results.

DR. AUSTIN A. HAYDEN asked Professor Russell if he routinely examined the larynx with a laryngeal mirror and a

tongue depressor at the same time. The patient usually holds his own tongue with a bit of gauze and no depressor is used.

He believed that a forward step had been made in the exact analysis of the location of deafness in the tone scale, and felt that the various electric hearing aids have acted largely as "shotgun prescriptions." They have magnified the entire tone range, without any regard for the particular pitch in which the patient heard the most, and for that reason a great deal of the effort of these instruments is lost. If the building up could be centered, as Professor Russell had pointed out, at that particular pitch in which the patient has hearing the results surely would be improved. He was glad to find that the work at the Ohio State University was in accord with the teaching of Dr. Kenyon.

Dr. Hayden thought the practical part of the science of phonetics could be summed up in a few words: That the removal of nasal and throat obstructions will help only a few of the speech defectives, and that when any operation is done on the nose and throat, with the idea of improving the speech, unless it be a definitely obstructive defect, one is treading on dangerous ground. Second, every otolaryngologist should equip himself with the name, address and telephone number of a speech specialist, for he has not the time or inclination to do this work properly himself. He believed the autofonofaryngoskop would be found to be the most useful of Professor Russell's instruments, for it allows not only the examining physician to view the interior of the larynx but permits the patient or a student to see the larynx, and it has the further possibility of having its image projected upon the screen. By making a small adjustment of this instrument the larynx and its adnexa can be seen in their normal size, whereas with the autoperilaryngoskop there is a much smaller image of a much larger field.

Professor G. Oscar Russell (closing), said that the cases of deafness which were cited showed an uneven loss which was greatest for the higher areas. If one amplifies enough for the subject to hear the high frequencies, the result will be an overpowering distortion of the lower. For this reason it would be necessary to create an apparatus which would balance the whole by amplifying the higher proportionately

more than the lower frequencies. That would be possible, but as instruments now run the result would be cumbersome to say the least.

He agreed with Dr. Hayden that the fonofaryngoskop is by far the best all-around laryngoskop of the five he has developed, for the reasons Dr. Hayden cited. It would be less expensive; injects neither spherical nor chromatic aberration, likely to result in lens systems; shows a strongly lighted area; more nearly natural in size; and where the autofonofaryngoskop is used, makes it possible for a large number of people to see the same thing at the same time.

On the other hand, he reiterated that the perilaryngoskop had many distinct advantages which he did not feel he needed to resummarize.

Regarding the hearing tests, he said he hoped he had made it clear that he was distinguishing sharply between the customary ones used by physicians in order to orient themselves in their diagnosis, and one whose diagnostic implications were primarily to guide the patient and his teacher. He did not mean to imply that the whisper and tuning fork tests did not have their advantages. But he made an earnest plea that some test be made which would give a sound spectrum analysis which would serve for pedagogical diagnostic purposes—one that would serve to guide the patient in his learning, relearning, or learning retention process; and give him a psychological reassurance which he felt was quite necessary in order to keep the individual from developing the well-known "deaf attitude of mind." The latter would have to be taken with some such instrument as the Western Electric 1-A or 2-A audiometer. (The 3-A or 4-A would not do.) If one considered the happiness which might be brought into the lives of these patients by such means, many times the expense of the apparatus would be justified. He hoped the otologists would come to agree that no patient should be released without such a chart to guide him as that he had just presented. If every doctor could not afford an audiometer, he suggested that (as for bacteriologic reports) the examination might be arranged for some central bureau of trained specialists.

## CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

*Meeting of Monday, April 1, 1929.*

DR. EDWIN MCGINNIS, THE PRESIDENT, IN THE CHAIR.

### PRESENTATION OF PATIENTS AND INSTRUMENTS.

DR. G. W. BOOT presented a patient who was admitted to Cook County Hospital on January 14, 1929, in a very drowsy condition. He had completed paralysis of both eyes for external motion, almost complete loss of internal motion but with vertical motion of the eyes preserved. He had some difficulty in naming objects. The left ear was discharging and a left mastoiditis was present. The left mastoid was exenterated, the left temporosphenoidal lobe exposed and one and a half ounces of pus evacuated from the temporosphenoidal lobe. The patient was very much emaciated at the time of operation. He had recovered much of his weight and there was almost perfect motion of the eyes in all directions.

DR. SAMUEL J. PEARLMAN presented a case of suppurative labyrinthitis following an acute exacerbation of a chronic otitis media with complicating symptoms of generalized meningitis ending in recovery.

R. L., white, male, aged 30, entered the Cook County Hospital on March 2, 1929. He gave a history of having had a chronic bilateral suppurative otitis media since early childhood, with an acute exacerbation in the left ear during the past four days, and presented on admission the picture of a full fledged meningitis which had apparently been present for about forty-eight hours. There was rigidity of the neck, bilateral Kernig sign, severe headache, nausea and vomiting. He had pain on pressure over the left mastoid. He had in addition a well marked nystagmus toward the right. Both ears were discharging freely, the left more than the right and the discharge was foul. There was no positive fistula test in either ear and the hearing was reduced to ad concham in the right ear and altogether gone in the left. Douching the left ear with hot water did not reverse the nystagmus.

Spinal puncture revealed a turbid spinal fluid under markedly increased pressure with positive Pandy and Ross-Jones reactions. The cell count was 29,000, chiefly leucocytes. Smears of the spinal fluid and cultures were negative at this time and at all other times. Wassermann was negative. A blood examination revealed a white count of 23,000 per cubic millimeter and the hemoglobin was 95 per cent. The spinal puncture was repeated practically daily and within nine days the cell count was reduced to 400 cells with a great improvement in all symptoms. The nystagmus, however, and the deafness persisted. On the sixteenth day after admission, when the patient was apparently quite well, he developed a facial paralysis on the left side. He had for a few days before this complained of a good deal of night pain in the region of the left mastoid. A radical mastoid operation was done at this time and revealed a small sequestrum in the horizontal semicircular canal, freely movable but not surrounded by granulations. This was not disturbed. Improvement was rapid and continuous after the operation.

In a recent discussion before the Scottish Society of Otology and Laryngology, Dr. J. S. Frazer summarized his opinion with regard to meningeal complications of suppurative ear conditions. He stated that those cases of meningitis in which no organisms were demonstrable in the spinal fluid did best; that where organisms could be demonstrated in smears from the spinal fluid, but could not be cultured, those patients sometimes recovered, but that where organisms could be demonstrated both in the smear and in cultures from the spinal fluid, the prognosis was almost uniformly fatal.

DISCUSSION.

DR. SAMUEL SALINGER said he saw both of these patients at the Cook County Hospital. In the case of the boy with the brain abscess there was considerable doubt as to whether there was an abscess, and if so, on which side, inasmuch as both ears were discharging with equal severity. They could find no focal sign except the bilateral rectus paresis, which was difficult to explain on the basis of a tumor in one part of the cerebrum. He thought the asphasia must have been transitory, or apparent only at times, for he had experienced

no difficulty in having the boy recognize different coins. The case demonstrated well how by persistence in questioning and frequent examination one can elicit a symptom on which the entire diagnosis may depend.

The case reported by Dr. Pearlman was also interesting, inasmuch as some authorities assert that when there is a suppurative labyrinthitis and meningitis the indications are to open the labyrinth, while others disagree with this dictum. In this case Dr. Salinger felt that as long as the boy's condition was improving it was proper to wait, as there was evidence that the process was walling off. He knew that the patient would require a mastoid operation, but thought best to wait until the labyrinthine symptoms cleared up. In the meantime the patient developed a facial paralysis, which to them was an indication that the bony necrosis was extending. A radical mastoid was promptly done, the facial paralysis disappeared and the recovery was complete.

Dr. J. Holinger asked if the dura was pulsating in the case of brain abscess, and whether there was much serous fluid.

Dr. Boot, replying, said the dura did not pulsate and he never had seen the dura pulsating in any brain abscess. The boy had over one hundred cells in the spinal fluid and a positive Kernig sign. In the last two months he had seen two such cases, one following a scarlet fever and the other an acute suppurative mastoiditis. Each had between 1800 and 1900 cells per cm. of spinal fluid.

SCIENTIFIC PROGRAM.

Dr. Isaac Abt addressed the Society on

**"Vienna of Yesterday and Today."**

Franz Hasslinger, Docent, Hajek Clinic, Vienna, delivered an address on

**"Laryngologic Problems."**

## AMERICAN COLLEGE OF SURGEONS

The American College of Surgeons will hold its nineteenth annual Clinical Congress in Chicago, October 14-18. Headquarters will be at the Stevens Hotel. An intensive program is being planned to make this home-coming event the greatest in the history of the College. The Hospital Standardization Conference will consist of morning and afternoon sessions on Monday to Thursday inclusive. There will be a series of clinical demonstrations given by: George W. Crile, Cleveland; John B. Deaver, Philadelphia; John M. T. Finney, Baltimore; Charles H. Mayo, Rochester, and others.

Monday evening's program will include an address of welcome by the chairman of the Chicago Committee on Arrangements, Dr. Herman L. Kretschmer; the address of the retiring president, Dr. Franklin H. Martin, Chicago; the inaugural address of the new president, Major General Merritte W. Ireland, Washington, D. C., and the John B. Murphy Oration in Surgery by Professor D. P. D. Wilkie of Edinburgh.

Among the foreign visitors will be: Dr. James Heyman of Stockholm, Dr. Thierry de Martel of Paris, Visconte Aguilar of Madrid and Mr. Herbert Tilley of London.

Tuesday, Wednesday and Thursday evening sessions will consist of scientific papers presented by surgeons from the United States, Canada and from abroad. The Annual Convocation of the College will be held on Friday evening. The Fellowship Address will be delivered by Dr. Glenn Frank, president of the University of Wisconsin. The annual meeting of the Governors and Fellows will be held Thursday afternoon, followed by symposia on cancer and bone sarcoma. An all-day session on Traumatic Surgery will be held on Friday, in which leaders in industry, labor, indemnity organizations and the medical profession will participate. A special program has been arranged that will be of interest to those whose practice is limited to surgery of the eye, ear, nose and throat.

A feature of the Congress will be the showing of surgical films that have been produced under the supervision and approved by the Board on Medical Motion Pictures of the College. New developments in color photography will be demonstrated. In addition to the commercial exhibits, there will be scientific exhibits by the departments of the College. A rate of one and one-half the regular one-way fare has been granted on railroads of the United States and Canada to those holding convention certificates.

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